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The Canadian Medical Association Journal

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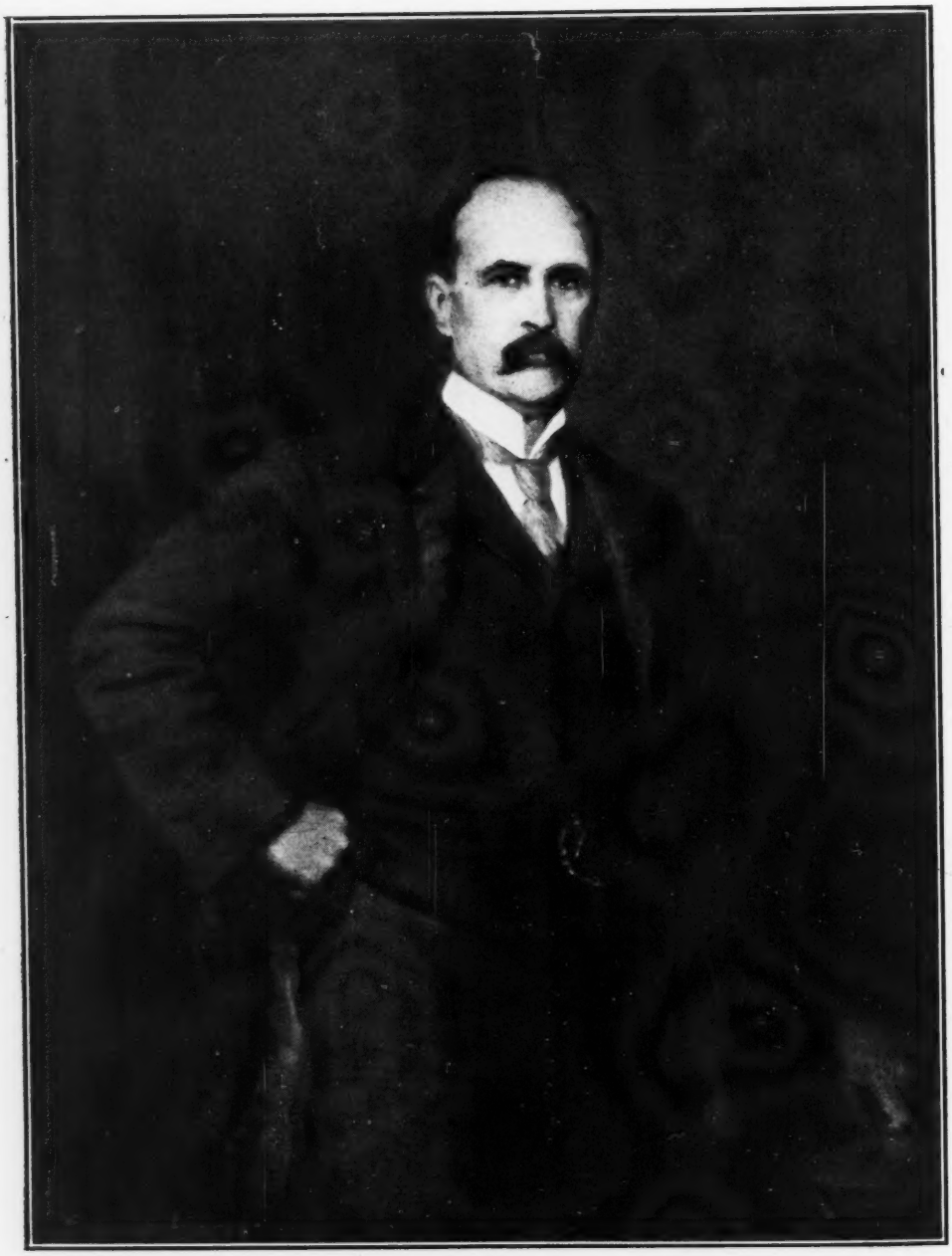

Wm Osler

The Canadian
Medical Association Journal

VOL. X. TORONTO, FEBRUARY, 1920 No. 2

SIR WILLIAM OSLER, BART., M.D., F.R.S., &c.

THE news received on December 30th, 1919, of the death of Sir William Osler, which had occurred on the previous afternoon at Oxford, England, threw a gloom over the whole medical profession of Canada. Sir William was as well known in Canada, his native country, as the premier.

Sir William Osler was born at Bond Head, Ontario, in 1849. His father was a Church of England clergyman, the Rev. F. L. Osler, M.A. (Cantab.). He was born on July 12th, hence was called William after William of Orange; he was the sixth son. Educated at Trinity College School, Toronto; Trinity College, Toronto; Toronto University and McGill University, Montreal, he graduated in medicine in 1872; he then went abroad for two years and studied in London, Vienna, and Berlin. On his return to Montreal in 1874 he was appointed to the Chair of the Institutes of Medicine in McGill University, made vacant by the resignation of Dr. Joseph Morley Drake, and held this Chair for ten years.

He was president of the Canadian Medical Association in 1884, and while residing in Canada was a great strength to the medical community, and did much to stimulate it and advance its interests. As a student he gave early promise of a great future; he was always investigating and trying out things, working independently with his microscope, an instrument which at that time was a rarity, and spending much time in the wards of the General Hospital and the post mortem room. He did not cram for examinations and took no high place in his class, but his graduation thesis on Pathological Anatomy was given a special prize and the specimens illustrating it were valuable additions to the museum.

Dr. Osler was called to Philadelphia to the Chair of Clinical Medicine in Pennsylvania University in 1884, and in 1889 the new

Johns Hopkins University and Hospital was forming a medical faculty, and the Chair of Medicine was offered to him. He accepted this post and remained there until 1904. His influence was the greatest in moulding the character and policy of this school, in which work he was assisted by Professor W. H. Welch and afterwards by Professor F. P. Mall. At Johns Hopkins he wrote his celebrated text-book on the "Practice of Medicine". This book achieved an immediate success which has never been surpassed. It is the most valuable single volume text-book in medicine ever written, and went through many editions. At a dinner given at the Waldorf Hotel, New York, in May, 1905, by the profession of Canada and the United States, six hundred medical men from all parts of America being present, Dr. J. C. Wilson said this about this Philadelphia period: "Not only by precept but by example has he been an uplifting influence in our professional life. . . . The source of that influence is to be sought not merely in his accomplishments as a physician, not in learning, not in his wisdom, not even in his well-balanced buoyant temperament, but in that basic principle which we all recognize but never can define, which for want of a descriptive name we call 'character'." Dr. W. H. Welch, in a speech at the same dinner to Osler, said: "His most striking contributions to the life at Johns Hopkins has been the interest which he has aroused among the students and the personal influence which has enabled him to bring out in them the best of their moral and intellectual points."

This is what the men of these two cities thought of him, and they voiced the opinion of the profession. The late Abraham Jacobi said at the same dinner: "The life-long work in which you invested your aims and ideals has ever been a labour of love and no hardship; you have not exerted yourself to earn thanks, and have expected none. . . . Your character and learning, your sound judgement and warm heart, your generosity and consistency, have gained thousands of friends. . . . There is nobody here or outside that coming near you has not been attracted, improved and inspired by you."

These are indeed words of great praise, but not too great, and are perfectly true; no one who ever met Osler could fail to be impressed by his personality and to feel that he was in the presence of a great man. In England he was much appreciated, not only as a physician but as a man and a force in the community; his unconventional ways and direct manner were a revelation to the rather exclusive, stand-off, university circles and his excursions into

literature were always welcomed by a large body of admirers. Whilst at McGill he instituted many reforms and innovations, and the dead bones of the faculty were brought to life again. He was a continual inspiration to the students and his colleagues. He made pathology an important part of the teaching and his vast experience in the post-mortem rooms and wards of the Montreal General Hospital was the basis of his great work in his "Practice of Medicine". Of course, all who knew him felt his personality; he was beloved by his students, chiefly because of his abundant sympathy, his naturalness and his vast fund of humour. His colleagues and medical friends were often the subjects of his practical jokes, which were always harmless and generally intensely amusing.

Whilst a physician to the hospital, he edited the first volume of the Montreal General Hospital reports, his own contribution consisting chiefly of the second part of his celebrated pathological reports, which occupied nearly one hundred pages. Dr. Maude Abbott, curator of the McGill Medical Museum, informs me that the specimens described in his pathological reports are still in the museum, having escaped the fire of 1907, and also that the wonderful specimens of endocarditis on which his Gulstonian Lectures of 1885 were founded, are still in good preservation.

When he left McGill for Pennsylvania, there was much regret but the spirit he had created and the influence he had exerted on the younger men remained. He was most suggestive as to the various lines of work he recommended to earnest students and many men owe much of their success to his stimulating personality and sound advice. He himself always said his life was much influenced by the three men to whom he dedicated his "Practice of Medicine". First, the Rev. W. A. Johnson, of Western Ontario; next, James Bovell, of the Toronto School of Medicine; and third, Robert Palmer Howard, professor of medicine in McGill University. The writer has often watched Osler unconsciously scribbling at meetings and afterwards looking at the blotter would find the name of James Bovell written all over it. He it was who first instructed Osler in the microscope, Dr. Howard encouraged and directed him in his pathological and clinical work and remained his warm friend until his death in 1889.

Sir William Osler was naturally of a religious temperament, and was much influenced by the Rev. Dr. Johnson. When a student at McGill, he was an ardent attendant at St. John's Church, a very high church indeed, under the care of the Rev. Mr. Wood. He was often seen at early service before breakfast. Had he lived

in the 12th or 13th centuries he would have been a monk and would no doubt have been a second Bishop Hugo.

As a clinical teacher, Osler was at his best; not only was he an acute diagnostician and a clear expositor, but he treated his hospital patients most kindly, as human beings and not as mere cases. His example was one which made a great impression on his students and the Osler tradition of gentleness and sympathy with patients was handed on.

Sir William's influence on medicine has been unique, for it has been exercised both on this continent and in Europe, so his death will be felt seriously over a wide araea. Not only has a great medical man gone from amongst us, but a great man. As an author on subjects not exclusively medical he was fairly prolific. His "Æquanimitas", 1904, "Counsels and Ideals," 1905, "An Alabama Student, and other Essays," 1908, were addresses delivered at one time or another on various ethical, historical, and biographical subjects. The Ingersoll Lecture, delivered at Harvard in 1904, created much interest and discussion. In it he said he would "rather be mistaken with Plato than be in the right with those who deny altogether the life after death". "A Way of Life," of 1914, was a charming essay. "Science and the War," 1915, was a lecture delivered before the Leeds medical students, and was rather a pessimistic address, though he looked forward to a time when "Nation shall not lift up sword against nation, neither shall they learn war any more". He also delivered last year a wonderful address before the Classical Association, of which he was president, which astonished everybody by his wide knowledge of literature. As a writer he had an excellent style, terse, to the point, every word telling, short sentences and no obscurity. He said what he wanted to say and then stopped—a rare quality in a writer. In his one volume on the "Practice of Medicine" there was more "meat" than in many systems. He loved books and libraries and was a great collector of rare editions and had a wide and accurate knowledge of many little known great books. His excursions into the historical side of medicine were numerous and instructive, and his biographical studies, such as that of "Linacre", were most accurate and valuable.

He was most human in all his ways and had high ideals but not unattainable ones. His influence in a medical community was always for peace, and against warring factions; under his direction enemies would lay down their weapons and become friends. He had a great love for little children and no one who has ever seen him in a children's ward could ever doubt it.

This obituary might suitably close with a quotation from his speech at the great dinner given him on his leaving the Johns Hopkins University for Oxford: "I have had three personal ideals, one to do the day's work well and not bother about to-morrow. . . . The second ideal has been to act the Golden Rule as far as in me lay toward my professional brethren and towards the patients committed to my care. And the third has been to cultivate such a spirit of equanimity as would enable me to bear success with humility, the affection of my friends without pride, and to be ready when the day of sorrow and grief came to meet it with courage befitting a man."

"I have loved no darkness,
Sophisticated no truth,
Nursed no delusion,
Allowed no fears."

Such was Osler, such were his ideals, and nobly did he live up to them.

Although far removed, especially in later years, he never forgot his Alma Mater; he was always endeavouring in one way or another to advance its interests. He was continually giving rare books to the library, or raising money for some object such as making and printing a catalogue of the museum, in which he took intense interest and which contained many of his own specimens. It is not a secret that he has left his magnificent library of rare books and incunabula to the McGill Medical Library with an endowment sufficient for its maintenance for all time.

Sir William Osler had received many honours from home and foreign lands; he had a hatful of LL.D.s., was honorary member of many foreign societies, was F.R.S. and F.R.C.P., London, and was made a Baronet in 1911.

Sir William was married in 1892 to Mrs. Samuel Gross, née Revere, of Boston, Mass., by whom he is survived. He had one son, who was the joy of his father's heart, having many fine literary tastes, but he, unfortunately, was killed in the war. This blow was a severe one and although Sir William bore up bravely, he never fully recovered from the shock. During the railway strike in England last autumn, Sir William had to take a long drive in bitter weather in a motor, and caught a severe cold; bronchopneumonia developed, then empyema with operation; a few days later he succumbed to sudden hæmorrhage.

F. J. SHEPHERD

THE INFLUENCE OF WILLIAM OSLER ON MEDICINE IN AMERICA

THERE will be few dissenters from the opinion that William Osler exercised a deeper and more far-reaching influence on American medicine than any other man. This applies both to his influence in a wide general sense and in a more restricted personal way. The former came particularly through his teaching and writings, with the demonstration which he gave in Baltimore of what a medical clinic might be. It is a matter of opinion whether his direct personal influence was not really the more important. No instance comes to mind of any one who had as strong an influence on so many men. There are many in America who have the Osler mark; in some it may be observed more plainly than in others. Count up the number of men who have worked under him and who occupy chairs in medical schools, and the extent of this influence is seen. One may ask how much this influence was exerted consciously. The answers would differ, but my own feeling is that much of it was unconscious and resulted from the nature of the man. He loved his fellowmen and to help came naturally to him. He gave of his best to those about him and they in turn felt that they could not do otherwise than give of their best.

It is perhaps well to describe some of the directions in which his influence was manifested under various headings, realizing that much of it cannot be classified under any label.

The Medical Clinic. As he has said himself, his great ambition was to establish a clinic in which the care of patients, the investigation of disease, and medical education and training could be carried on. How well he succeeded is demonstrated in Baltimore, where in the Johns Hopkins Hospital he developed ideas which were new to this country. The clinic he established there was the best of its time, and his methods have been widely copied. One important point was the system of a permanent resident staff. Men entered the department as assistants with the object of a training in internal medicine which was to extend over a period of years. The first assistant in the clinic—known as the resident physician—came to the position after years of training as an assistant. This resulted in the head of the department being able to hand over part of the routine administration to his staff and so save himself from much detail work. These men aided in carrying on investigations at the same time that they were learning medicine and how to teach.

The influence of such a clinic is important and is exerted on the community as well as on the profession. In the last estimate perhaps the men who trained under this system carry more widely than any other method could the influence of the directing mind. Every man trained by William Osler is extending his influence to a greater or less extent, and these circles of influence are always growing wider.

Education. It is perhaps difficult for Canadians to realize how much change has been brought about in medical education in the United States in the last twenty-five years, because clinical teaching in Canada at that time was so far ahead of that south of the border. Much of what we have to-day in clinical teaching we owe to Edinburgh, just as London owed it to Edinburgh about one hundred years ago. The close association between Edinburgh and the Canadian schools resulted in the introduction of bedside instruction into the latter at an early date. At the time of the opening of the Johns Hopkins Medical School there were few medical schools in the United States in which the students worked in the wards. One of the things which William Osler accomplished was to prove that the medical student should be regarded as an integral part of the hospital organization. I believe that few things gave him more satisfaction than this, and its importance in medical education cannot be over-estimated.

In teaching, he did away with the didactic lecture; there were none in medicine in the Johns Hopkins School. Whether the didactic lecture has a place in medical education is a matter about which there are different opinions, but his practice emphasized the fact that the best place to learn medicine is at the bedside. The influence of this change has been very evident in American teaching.

Medical Societies. William Osler was a strong supporter of the societies to which he belonged and most regular in his attendance. This was true in each city in which he lived. In the local hospital and city societies, in the state and national societies it was the same. He usually gave more than he got, but it made no difference to him. On one occasion, on the day of a meeting of the Hopkins Medical Society for which the programme was not particularly interesting, he asked a young physician if he was coming to the meeting. "No," was the reply, "I do not think I would get anything out of that programme." "Do you think I am going for what I can get out of it," was Sir William's quiet reply.

It was a delight to hear the clear presentations which he made himself and to have him sum up at the end of a muddled discussion.

There was always the word of encouragement for the young man but a quiet thrust at anything which was not genuine or superficial. He was always encouraging his assistants and students to present cases and read papers.

He had much to do with the development of the Medical and Chirurgical Faculty of Maryland (the state society) and with the growth of its library. He was especially active in the fight against tuberculosis and took a leading part in the organization and the National Tuberculosis Association.

Books and Libraries. Books were his avocation through most of his life, and had he been spared for more years they might have become his vocation. Libraries were always his delight, and no long time went by without some library being remembered by him. In Oxford particularly he always seemed to have some book which was going to McGill, or to the College of Physicians of Philadelphia or Baltimore. He did much to interest the profession in books and libraries, and more especially in the historical side of our art. He emphasized the value of an historical perspective and excited the interest of the profession to acquire it. But his influence was not confined to medical litarature. To physicians and students he showed the way to a knowledge of general literature. For example, how many owe their introduction to the works of Sir Thomas Browne, Montaigne and Robert Burton to William Osler?

The Community. His influence was strongly exerted for measures which had to do with public health and his opinion carried great weight with his fellow citizens. In Baltimore he aided materially in the campaign to convince the community that a pure water supply and a proper sewage system were necessary. In the same way he did much to aid the campaign against malaria.

Treatment. He always emphasized the importance of accurate diagnosis as a preliminary to proper treatment, not failing to point out that sometimes symptomatic treatment may be all that is possible. If this is to be a "Therapeutic Nihilist", then he was one, but few of us would agree to this. Of the importance of proper and energetic treatment no man was a stronger advocate, but with those whose chief idea of treatment was embraced in a random polypharmacy, he had no sympathy, and some such men were fond of attacking his methods of treatment. He did much to reduce the amount of unnecessary drugging. In his teaching, emphasis was given to the need of considering the patient as an individual and not directing attention to one organ. The importance of the general mode of life, the surroundings, the influence of

worry and strain and the need of removing these if possible, and the use of cheerfulness, bulked largely in his therapy. But he never minimized the usefulness of drugs properly employed.

There are less tangible ways in which his influence was marked. Who can sum up the effect of his idealism, his kindness, his sympathy, and his generosity? Every man who came under his influence absorbed these to some degree and passed them on to others. His charity to all men was perhaps the personal characteristic which impressed many of his pupils the most. And so we leave him. The mortal body has gone, but the spirit and influence of William Osler remain.

THOMAS MCCRAE

THE PATHOLOGICAL COLLECTIONS OF THE LATE SIR WILLIAM OSLER

AT this time, when Canada is mourning the loss of one of the greatest of her sons, and the Canadian profession its most beloved and distinguished member, it is of interest to know that this country possesses, in the form of the large and valuable pathological collections made by Sir William Osler in the years 1877 to 1884, a unique memorial of the early formative years of his professional life. This collection was made by him during the years he acted as pathologist to the Montreal General Hospital, and they are housed, with full records pertaining to them, in the Medical Museum of McGill University. They include the fine series of specimens of malignant endocarditis, on which he based his Gulstonian lectures on this subject; also some thirty specimens of aneurisms on which his work on this subject and on angina pectoris was based; a fine series of cardiac anomalies, cardiac thrombi, gastric ulcer and carcinoma, coal-miner's and stone-mason's lung and many other conditions. All are referred to repeatedly both in his "Practice of Medicine", and in his "System" articles. Each specimen has been carefully dissected by him to show the lesion, and each is of pathological as well as of biographic and historic interest to-day. The reports of his autopsies, done at the Montreal General Hospital on these and other cases, fill three large volumes which are written entirely in his own clear flowing hand, and give evidence, to an extraordinary degree, of his powers of clear diction and minute observation. These reports, with the specimens to which they refer, undoubtedly present, in visible and tangible form, the first stepping-stones in a great career.

MAUDE E. ABBOTT

FOCAL INFECTIONS

ILEAL AND COLONIC STASIS

By G. E. Armstrong

Surgeon-in-Chief, Royal Victoria Hospital, Montreal

DURING the past ten or fifteen years the attention of the profession has been directed to auto-infection. The question has been approached from different angles by different observers. Many conditions, the causes of which were little understood, as shown by the varied and different theories advanced concerning their nature, are now shown to be due to a chronic toxæmia, the toxins being manufactured in the patient's body. Our knowledge of these conditions, so far as at present understood, has been obtained in the clinical research departments of our hospitals. Chemistry, physiology, anatomy and biology may, we hope, be able to further clarify our views in the near future.

The list of ailments ascribed to auto-infection has been added to by various writers, until it has become notable for its length and variety. It has included so-called rheumatism, heart disorders, neurasthenia, goitre, chronic mastitis, lassitude and a host of other diseases and variations from the normal.

"Alimentary toxæmia," an objectional term, has come into rather general use. The term is unscientific. It indicates the port of entrance rather than the nature of the poison. The simplest alimentary toxæmia is that due to pyorrhœa alveolaris which may be secondary to a toxæmia arising in some distant part, or again micro-organisms may be carried from the mouth to the stomach, and if not destroyed there, on into the intestines. The necessity of a careful examination of the teeth, mouth, and pharynx in all cases where auto-infection is suspected is now pretty generally recognized by the profession.

In the years 1902 and 1903, three men—an anatomist (Barclay Smith), a bacteriologist (Metchnikoff), and a surgeon (Sir Arbuthnot Lane), came independently and by a different train of reasoning to the same conclusion, which was, that, so far as man is concerned, the great intestine is not only a useless but a pernicious structure.

From that date to the present the attention of the profession has been directed to focal infection, and more particularly, perhaps, to that arising in the large gut, by the writings of one of the most interesting and original of living surgeons, Sir Arbuthnot Lane, of Guy's Hospital, London.

The idea that proper action of the bowels is conducive to health and a sense of well-being is a very old one, although it is said that the doctrine of intestinal auto-intoxication originated with Bouchard. Certainly there is a widespread belief that many diseases as well as loss of reparative power depend upon absorption of poisons from the intestines, either introduced with the food or formed from it by the fermentations or putrefactive processes that take place in the alimentary canal.

The foregut, including the posterior wall of the pharynx, the œsophagus, stomach and duodenum down to a point below the entrance of the common duct, together with the liver and pancreas, are concerned with the preparation of food. The only absorption being a small quantity of spirits from the stomach.

Absorption is the function of the mid-gut, which functionally extends to about the hepatic flexure. We get our water from the cæcum and ascending colon. Nevertheless it is shown by the comparative good health of people from whom the colon has been removed that we can get sufficient water when the colon is absent. This fact does not, of course, prove that the large gut is either a pernicious or useless organ. It is well known that man can live in good health without a colon, but it has never been demonstrated that man enjoys better health without than with a healthy colon. Sufficient evidence has accumulated to justify the statement that man is in many instances in better health without a colon than with a diseased colon.

Sir Arbuthnot Lane attributes the failure of the large gut to functionate to mechanical displacement. The mechanical displacement he ascribes to weakness of the normal supporting tissues, that have a great strain put upon them by our erect posture.

The natural support of the contents of the abdomen are the abdominal and pelvic muscles. The hardy Italian navvy does not suffer from enteroptosis. The question has been raised as to whether the low-lying position of the stomach and intestines is really obstructive, in the sense of causing material delay in the passage of their contents.

Before 1895, we had no accurate information regarding the rate of progression of food along the alimentary tract. We now

know, thanks to the discovery and use of the Roentgen rays, that food is normally delayed at certain points of the alimentary canal. The anatomical arrangements at the end of the œsophagus, at the junction of the stomach and duodenum, at the situation of the Ochsner's muscle in the duodenum just below the entrance of the common duct, at the ileocæcal junction, at the commencement of the transverse colon, at the juncture of the rectum and colon and at the sphincter of the rectum, cause a delay that may be considered normal.

The view that an abnormal delay amounting to stasis is due to bends that result from ptosis is challenged by the experience of abdominal surgeons and pathologists. They say, quite truly, that ordinary bends and considerable narrowings of the small and large intestine may exist without being really or materially obstructive. That above these bends and narrowings, unless they are extreme, there is no hypertrophy of the muscular coats of the gut. On this point it must be remembered that not at all infrequently the cæcum and ascending colon are found greatly thinned and dilated.

A new view of the question of intestinal stasis has recently been proposed by Arthur Keith, curator of the museum of the Royal College of Surgeons, London, and it is a most attractive theory. It relates to the nerve supply of the musculature of the stomach and the small and large intestine. When we become physically tired from muscular exertion, it is not the muscles that are fatigued, but the nervous excitant or stimulus is lowered. Restore the nervous energy and the muscles are quite ready to continue their work. The musculature of the intestines is the sole propelling power. Increased strain may be put upon it by the mechanical conditions that obtain in enteroptosis. There may occur a fibosis of the muscular coat, and Keith's work certainly suggests very forcibly that the nerve supply to the œsophageal, gastric and intestinal musculature may be so altered that the deficiency or lowered nervous stimulus may contribute to abnormal delay and stasis.

Physiologists teach us that all the non-striated muscles of the body, the musculature of the alimentary tract, of the bladder uterus and ureter have the same property as cardiac muscle, a power of rhythmical contraction which it continues to exercise as long as it is alive.

Another most interesting observation was made by Bayliss and Starling, viz.: that every piece of bowel possessed a double rhythm or beat, a rapid one in which the contraction waves arose

at the rate of ten to twenty a minute, and a slow and concurrent one in which the contraction waves arose at the rate of one or two a minute. They further showed that the slow beats gave rise to true peristaltic waves. The more rapid and slight rhythm was concerned with division and mixing of the intestinal contents. Mall suggests that the more rapid and rhythmical contraction is directly concerned in the maintenance of the portal circulation. As possibly throwing some light on the ætiology of appendicitis, it is to be noted that the appendix is possessed of a peculiar arrangement of the myenteric plexus and evidently possesses a peculiar mechanism for becoming filled and emptied and has a double rhythm of the same kind as found in the alimentary tract.

Keith presumes that the myenteric plexus represents in the intestine a system which corresponds to the nodal and conducting system of the heart. He has sought to distinguish four rhythmical zones in the intestine: a duodenal, jejuno-iliac, a proximal colic and a distal colic. There are likely two others, a gastric and œsophageal. He thinks that the nodal tissue around the cardia acts as the pacemaker of the stomach and that the nodal tissue found round the ileo-colic junction acts as the pacemaker of the colon, in the same way that the nodal tissue in the sinoauricular and auriculoventricular grooves acts as the pacemaker of the heart.

He found that "a series of sections through the ileo-cæcal junction of the rat's bowel revealed a collar of peculiar tissue, with two extensions which passed into the anterior and posterior walls of the cæcum. In the collar or ring could be recognized nerve cells and nerve fibres, but there was also a third element. Numerous branching cells, not connective tissue in nature, with processes which united with muscle cells, on the one hand, and with the processes from true ganglion cells on the other." He regards these intermediate cells as a possible representation of the nodal tissue of the heart.

We have here two theories to account for abnormal delay or stasis in the terminal portion of the ileum and proximal colon. The theory of insufficient, defective or abnormal nervous innervation of the musculature of the intestine has much to commend it. It suggests two questions. How far is it due to diet and how far to the strenuous life of our age?

Our meat consumption has increased very much during the past hundred years. Meat when not properly digested undergoes putrefaction, and toxic matters are formed which may quite probably interfere with the nerve cells, and lower their efficiency. Then

again our bread, the staff of life, is so far refined that it resembles a bill-poster's paste. The stimulating or irritating outer layers of the wheat are almost altogether removed. If present they would stimulate peristalsis.

The strenuous life of our day tends to nerve exhaustion. As Oliver Wendell Holmes puts it:

> "We have found our place
> Just in the focus of a nervous race
> Fretful to change and rabid to discuss,
> Full of excitements, always in a fuss.
> Think of the patriarchs: then compare as men
> These lean checked maniacs of the tongue and pen."

The weak link in the chain is that we have no radiographs of the ileo-colic regions in the patriarchs.

The cæcocolon which permits passage of toxins arising either from bacteria or from the gut wall itself is the offending organ. It is biologically younger than the descending colon and more prone to change and variation. A very interesting paper by Draper (*Annals of Surgery*, May, 1918) advocates "developmental reconstruction of the colon," *i.e.*, removal of 10 cm. of the terminal end of the ileum andiof the cæcum, and ascending colon to the neighbourhood of the right colic artery.

I have now performed this operation in six cases, removing the lower end of the colon and cæcocolon in one mass.

In two cases the end of the colon was closed and the end of the ileum anastomosed with the side of the colon. In four cases I did an end to end anastomosis as advocated by Balfour, at the Mayo clinic. I have not had any mortality; in fact, the operation is not particularly difficult and the convalescence remarkably smooth, the temperature seldom rising above 99°.

The improvement that followed operation was very marked in all cases. There was no mortality. I shall report on the later condition of these patients at some future time.

CHRONIC INTESTINAL STASIS

By D. T. Smith

Ottawa

IN discussing the subject of chronic intestinal stasis, only the most important phases can be touched upon in a short paper for purposes of discussion, therefore we shall accept Mr. Lane's premises.

The definition of chronic intestinal stasis is somewhat confusing with chronic constipation. The definition of constipation which has formerly been accepted as adequate does not describe what we know now as chronic intestinal stasis.

Constipation is usually considered to involve the large bowel, particularly in its lower portion; it results as a rule from an improper diet, an insufficient fluid intake, a lack of exercise, or a general atonic condition of the body tissues or a combination of two or more of these functions. The condition may, and often does supervene, even in marked degree, when the lumen of the bowel is entirely free from angulation, kinks, and other obstructive abnormalities. Furthermore, constipation may exist to a very pronounced degree even in the intractable form known as obstipation, and yet the patient may suffer very little from the effects of absorption of the retained material and its toxins.

In chronic intestinal stasis, on the other hand, while the factors which produce constipation may be operative, others involved are definitely demonstrable by diagnostic means at our command. In the first place, according to Lane's theory, the evolution of man from the all-fours posture of his progenitors of field and forest, results in a general tendency to visceroptosis. The dropping of the abdominal organs gives rise to stress and strain upon the mesentery and its attachments. Nature attempts to offset this strain by the formation of practically bloodless evolutionary bands. These bands develop with unequal strength in different parts and the result is unequal support. The bowel is held up firmly at some points while it is allowed to sag at others. Angulation or kinking

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at the point of support follows this abdominal fixation at a given point in the length of the intestine, while a dropping of the tube on either side narrows the lumen of the gut to a greater or lesser degree and thus interferes with the passage of its contents. The immediate result of this alteration in the drainage scheme is such a slowing in the passage of the food along the alimentary canal that an excess of toxic matter is formed especially in the small intestine; in other words, the condition of stasis supervenes. Inasmuch as the factors which lead to this are not transitory but permanent unless corrected, the condition becomes chronic and hence we have chronic intestinal stasis.

The blood stream in such a case surcharged with the toxin taken up from the retained or residual fæcal contents of the intestine conveys to the transforming and excretory organs larger quantities of these poisons than they can eliminate. All the tissues of the body then become supplied with blood ladened with toxins, they are improperly nourished, as a consequence, they deteriorate and are soon unable to offer the accustomed and proper resistance to infection and disease. The pros and cons of the matter are being thoroughly threshed in the wheels of experience and controversy. (Pathologists, radiologists, gastroentrologists, and surgeons are taking part in the discussion of this important and seemingly far-reaching chronic intestinal stasis and its treatment.) If we thoughtfully consider this disease we must accept Sir Arbuthnot Lane's views and contentions; as we know Lane contends that chronic intestinal stasis is the ground-work upon which the superstructure of many diseases is placed, although we find the seventeen symptoms and nine diseases enumerated by him have been pronounced by Dr. Adami to be a horrible jumble. Perhaps so, yet there is undoubtedly enough in Lane's contentions along this line to warrant serious attention rather than sneering and jocular comment.

In the light of latter day finding it is unwise to reject any serious suggestions from a conscientious, scientific worker who has said for example to have been the first to reset a rib for empyema, the first to introduce saline solution into human blood vessels, the first to tie off the internal jugular vein for sinus thrombosis, the first to plate bone, the first to operate on cleft palate and hare lip at the same time, and the first to study chronic intestinal stasis as an entity, to short circuit and remove the colon for correction of the condition; therefore we must take Lane's theory for purposes of discussion, and also that of Alfred C. Jordan, of London, who has been associated with Lane in fluoroscopic and *x*-ray work.

What then are the clinical symptoms of chronic intestinal stasis? They may be enumerated roughly in the following order: (1) Pain or discomfort usually referred to the region of the duodenum and stomach, but also to portions of the large intestine; (2) Gastric discomfort, nausea and occasional vomiting, resulting from obstruction to the outlet of the stomach in consequence of ulcer or cicatrization of the pylorus or duodenum, or constructing bands about the duodenum in the neighbourhood of the pylorus. These symptoms may be classified under the ordinary category of indigestion. (3) Various symptoms which may be catalogued under the term of intoxication, which Lane has described as flooding the liver with a quantity of toxic material from the stomach, duodenum and small intestines; in excess of what the liver, kidneys and skin are able to deal with, these symptoms vary according to the susceptibility of the individual. Under this head may be grouped a set of individual symptoms and physical signs such as the blotched appearance of the skin which is cold and clammy, especially over the extremities, cold perspiration of an offensive odour, the loss of fat, and a lumpy condition of the breasts. Thyroidism in some cases may exist with tenderness over the ilium and mental torpor. In fact, the entire symptomatology usually described under auto-intoxication, headache, melancholia, inability to sleep, unpleasant dreams, and occasionally persistent diarrhœa, come under this general classification of symptoms.

The patients who present a sufficient number of these signs and symptoms briefly catalogued above warrant a tentative diagnosis of chronic intestinal stasis, and should be safely guarded in every way. The Wassermann test should be done where there is a probability of syphilis. When the clinical examination points definitely to stasis, confirmed by *x*-ray examination the abdomen should be opened. The treatment should be considered under three general rules. In the first group are cases in which by preventive measures definite conditions of stasis may be obviated. In the mid-group are mild cases in which by preventive measures and by modern surgical procedures such as cutting bands, replacing hollow organs, changing angles, the severe degrees of stasis are forestalled and the necessity of more radical surgical procedures may be prevented. The end group are advanced cases in which, despite preventive treatment or because of insufficient treatment, the condition progresses to the degree of stasis which requires a more radical surgical procedure, such as short circuiting, with colectomy. The surgeon who is called upon to explore the abdomen in every case such as this approaches more or less of a mystery. How-

ever careful the examination may be, the diagnosis is somewhat uncertain until the abdomen is opened and it is important therefore that the operator bear in mind that rare conditions may be found where ordinary ones are expected, and that he be able to cope with whatever state of affairs may be encountered.

The Results of Auto-Intoxication on the Tissues

I will now pass on to consider the consequences which result from absorption chiefly from the stomach and small intestines, of the products of bacteria or chemical changes which exist in abnormal quantity in the material from which the food supply of the individual is obtained. The infection of food supply is consequent on the damming back of the material in the small intestines and stomach. It would appear that very little poisonous material in the small intestine is absorbed, unless there be a super added infection of its mucous membrane. This is demonstrated very clearly by the study of the congenital dilatation of the colon. Very little is known of the material that is absorbed into the circulation in simple stasis, but if it is presented in excess of what can be dealt with by the liver, kidneys, and skin, certain very defined symptoms arise which are clearly due to the presence of some abnormal poison or deleterious matter in the blood.

Professor Arthur Keith has done a great deal of original work on the functions of the large bowel; the following quotation from a lecture delivered at the Royal College of Surgeons and published in the *British Medical Journal*, December 7th, 1912, has an important bearing here. The title of the paper is "The Functional Nature of the Cæcum and the Appendix". "Every year the opinion gains ground that the great bowel from the appendix to the rectum so far as man is concerned is a useless and dangerous structure. Exactly ten years ago, October, 1906, Dr. Barker Smith, of Cambridge, gave a clear expression of this new conception. Dr. James McKenzie has recognized and described the heart changes consequent on auto-intoxication so thoroughly in his work on the subject claiming the normal degree of blood pressure is soon restored to the patient when the large bowel is excluded by operation. He employed the term 'ex-disease' for the reason that he did not know the nature of the complaint; but one thing he did realize from cultivation of ileal-chyme obtained during life from the subject of constipation, almost invariably revealed the presence of numerous micro-organisms and that the living bacteria presented in one cubic centimetre of fæcal contents are many thousand-fold more numerous

than those in a cubic centimetre of ileal-chyme in the intestinal tube. The organisms most commonly found are bacillus coli, streptococci, often alive. Other organisms sometimes found are staphylococcus citreus, *e.g.*, in Still's disease, and bacillus aminophilus."

We will now consider the conditions of so-called diseases which arise in the individual because of the lowered vitality of the tissues by the presence of toxins or poison in the blood. In toxæmia the organisms which exist normally in the mouth are able to secure a foothold in the individual between the teeth and gums and manifest their presence as infective or inflammatory process. As a quantity of organisms are grown in these nurseries, the absorption of their products assist in the general process of depreciation. Many observers are inclined to regard these secondary foci as primary, but a little consideration will show that removal of the teeth has not relieved the symptoms of intestinal stasis, which was the primary factor. However bad the condition of infection of the gums may be at the time of short circuiting, an immediate and extraordinary improvement in this disease follows on the clearing up of the small intestine. To put the matter briefly, the upper alimentary tract is specialized for aseptic absorption of food and the colon for bacterial destruction of the residue. The indirect changes are those that result from the lower resisting power of the tissue to the invasion of organisms produced by auto-intoxication and can hardly be separated abruptly from all those conditions described as being caused directly by it.

First, the most obvious are pyorrhœa alveolaris; second, tubercular infection when not produced by direct inoculation; third, rheumatoid arthritis. This, like tubercle, cannot develop in the presence of effective drainage of the gastro-intestinal tract. Fourth, infection of the genito-urinary tract, either directly or indirectly through the blood streams, by organisms other than tubercle, producing nephritis, cystitis, pyelitis, endometritis and salpingitis. Fifth, development of changes in the thyroid gland, whether as adenomatous tumours or general enlargement of the thyroid gland or exophthalmic goitre. Sixth, Still's disease; seventh, infection of the skin of a pustular nature; eighth, infection of the large intestine by organisms which produces several varieties of mucous and ulcerative colitis; ninth, ulcerative endocarditis.

I have chosen then merely a few obvious typical indirect results of the auto-intoxication of chronic intestinal stasis. The point of practical interest in conclusion with these indirect results of stasis is that the resisting power of the tissue of the body is such

that they can destroy the organism or the poison which produced the condition if not too advanced or if cancer has not developed.

In retard to radiographic finding, I wish to refer to an article published by Dr. James T. Casem, Battle Creek, Michigan, where he claims that with symptoms formerly attributed to prolapsus we are now finding more tangible lesions such as chronic appendicile, disease constricting membranous bands along the ascending colon, veils about the hepatic flexure, adhesions attending gall bladder disease, torsion and twisting od the transverse colon, due to omental adhesions and especially spasticity and adhesions in the iliac and pelvic colon.

It is important to repeat here an expression by Dr. Alfred C. Jordan, a noted authority on *x*-ray work, who distinctly emphasizes the fact that at the bottom of the case disease can generally be revealed by a complete radiological investigation of the alimentary system, and it is just this contention which should stimulate radiologists to make careful and painstaking investigations along the lines laid down by Lane.

To Dr. Bainbridge I am greatly indebted for he has given me a clear view of the essential facts of chronic intestinal stasis. When in New York last those cases he presented and operated on were most convincing. I saw many cases, each day, some of them before operation at the time of surgical treatment, witnessed his masterly technique, noted the history of the convalescent period, talked with staff and nurses and patients, and from this limited experience I believe chronic intestinal stasis deserves our most serious consideration.

Medical treatment. Aside from tonics and other supporting measures, little medication is necessary; in some cases, however, it may be necessary to have recourse to laxatives and exercise in order to stimulate the peristalsis. From the surgeon's point the treatment consists of facilitating the passage of material through the several portions of the gastro-intestinal tract, aided by the use of liquid paraffin, the application of some spring support to the lower abdomen, massage, and the avoidance of such proteid foods as poison the tissues. When these methods fail, resort must be had to operative interference. The essential object of such operative treatment is to facilitate the effluent from the ileum and so to remove at once from the drainage scheme the stagnating material from which toxins are chiefly supplied.

I have tried to bring forward enough evidence to make the subject clear and I trust also to convince you that chronic intestinal stasis is a subject that merits your careful and thoughtful study.

THE WINNIPEG EPIDEMIC OF ENCEPHALITIS LETHARGICA

By William Boyd, M.D.

Winnipeg

WHETHER to the internist, the neurologist, the pathologist, or the epidemiologist lethargic encephalitis is a disease of absorbing interest. The geographic distribution and general epidemic behaviour of this mysterious disease are baffling to a degree. In distinction to influenza which swept over Europe with express trains, crossing the Atlantic with fast liners, and wandering through Asia with camel caravans, epidemic encephalitis appears now here, now there, descending on the startled community like a bolt from the blue. In this it bears a close resemblance to acute poliomyelitis, a disease much given to making sudden appearances, assuming the proportions of a small epidemic, and then vanishing, only to reappear at some distant point. The explanation in both cases is probably the same, namely, that the abortive cases and carriers greatly exceed in number the typical examples of the disease, so that the infection is much more widely distributed than might be supposed.

The malady made its début in Vienna in the winter of 1916-17, where it was first described by von Economo in April, 1919, who christened it "encephalitis lethargica". The first case noted in England occurred in February, 1918, and during the following four months about two hundred and thirty cases were reported in London and the provinces. In the United States the first cases were described by Bassoe in the *Journal A. M. A.*, March 1st, 1919. During the present year localized epidemics of small proportions have occurred in New York, Chicago, and other large centres, but in no case has the disease assumed pandemic proportions.

The disease made its appearance in Winnipeg in the last week of October, and during the succeeding weeks fresh cases presented themselves almost every day. In not a single instance could any connection be traced between the different cases, and never more

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than a single case occurred in any one household. Several of the cases were sent in from isolated farms in the surrounding country.

The disease is a febrile one, involving the grey matter of the brain stem, and presenting symptoms usually so characteristic that a diagnosis can readily be made. Many cases are met with, however, in which one or more of the usual symptoms are absent, and many examples of what may be called "formes frustes" occur, in which no definite diagnosis can be made, so transient and fleeting are the symptoms, but which excite a strong suspicion that they should be grouped under the same heading.

For clinical purposes the cases may be divided into (1) those with general but no localizing symptoms; (2) those with both general and localizing symptoms. The localizing signs may appear early in the disease or not until later. The general symptoms are those of a general infection, and there may be one or more of the following:—fever, lassitude, headache, pain in the back, furred tongue, loss of appetite, constipation, conjunctivitis, and urinary changes such as the presence of albumen and blood. In a number of cases constipation was a marked feature; indeed, in one instance the first symptom noticed by the patient was the impossibility of getting the bowels opened. In others, the foul breath and thickly coated tongue furnish unmistakable evidence of a general gastro-intestinal disturbance. In one or two cases a severe degree of conjunctivitis has been present.

The importance of these indications of a general infection lies in the support which they give to the view presently to be advanced that we have been mistaken in regarding encephalitis lethargica as a purely cerebral infection with accompanying fever, etc., such as might accompany a cerebral abscess. It is rather a systemic infection with special localization in the brain, an infection which may involve the parenchymatous organs to a greater or less extent, and which perhaps in some cases may spare the brain, thus giving rise to atypical forms of the disease which could hardly be diagnosed in the present state of our knowledge. Such a conception would agree with that now held with regard to cerebrospinal meningitis, poliomyelitis, and other infections of the central nervous system.

Symptoms

The *onset* varies greatly in suddenness. Sometimes there is a distinct prodromal period, a period between the time when the patient first feels that he is not quite well and that at which characteristic features of the disease make their appearance. This period may vary from one to ten days or longer. As an example of

a long prodromal period, the case may be cited of a woman who had been suffering from a moderate degree of headache and feeling out of sorts for about three weeks. At the end of that period she developed internal strabismus due to paralysis of the sixth nerve. When seen in conjunction with Drs. Bruce Hill and Burridge, the diagnosis appeared to be one of cerebral tumour or cerebrospinal syphilis. The spinal fluid was normal. Five days later she became very lethargic and developed bilateral facial paralysis. The case was undoubtedly one of lethargic encephalitis, but the onset was so slow as to suggest cerebral tumour.

On the other hand, the onset may be so sudden that one hesitates from making a diagnosis. In two of our cases it was apoplectiform in nature, the patient falling to the ground, and in one instance becoming unconscious. As a rule, two or three days elapsed before the characteristic symptoms made their appearance, but the period varied greatly in the same mamner as did all the manifestations of this remarkable disease.

Age. The age varied between the extremes of eighteen months and seventy-two years.

Fever. Epidemic encephalitis is essentially a febrile disease, but the temperature is very variable; there may be no fever during the early stages of the disease, and in one or two of our cases the temperature went up only when the patient had become dangerously ill. It is well that this should be recognized, for the fact that the patient may have a normal temperature may readily lead to mistakes in diagnosis. The temperature as a rule varies between 100° and 102°, but towards the end of the illness hyperpyrexia may occur. The temperature cannot be taken as a certain indication as to the progress of the case. In the fatal cases, it is true, it seldom or never came down to normal. In some cases, however, in which the lethargy and paralysis cleared up in a day or two, the temperature remained above normal for a week or more. This peculiarity can be understood if we regard lethargic encephalitis as a systemic infection specially localized in the central nervous system, but involving other organs as well. This has been proved to be the case in poliomyelitis, and post-mortem evidence in some of our own cases makes it practically certain that the same is true in lethargic encephalitis.

Lethargy is so characteristic a feature that it has given the name to the disease, and yet it is not present in every case. It is possible that both the lethargy and asthenia are due, not so much to the causal virus, as to the site of election in the brain. That site of election is the mesencephalon in general and the peri-aque-

ductal region in particular, and it is a well-known fact that lesions in this part of the brain are often associated with marked degrees of somnolence. In this connection it is of interest to note that two structures come into intimate relationship with the mid-brain, the pineal body on the dorsal aspect, and the pituitary body on the ventral, the infundibulum of the latter arising from the floor of the third ventricle. Disturbances of the pituitary often give rise to conditions of lethargy, as in Fröhlich's syndrome. Those of the pineal may be associated with lethargy and adiposity, together with various forms of ophthalmoplegia.

In some of our cases lethargy associated with a rise of temperature was the only symptom. In one or two it was absent, and yet the diagnosis was not in doubt. These are probably cases in which the brunt of the attack falls upon the cerebral cortex rather than on the brain stem. One man, for instance, showed symptoms of maniacal excitement throughout the illness and at no time manifested any trace of lethargy, but the brain showed the typical lesions of the disease. The lethargy is usually associated with great drowsiness, and the patient may pass much of the day plunged in deep sleep. Curiously enough, insomnia at night is not infrequent, and there may be definite delirium. However deep the lethargy, however, it was almost always possible to arouse the patient, and remarkably clear answers to questions could be obtained. The patient would then close his eyes and relapse into his former state. One case was aroused for purposes of lumbar puncture, but when the needle had been introduced he at once sank into lethargy again.

Asthenia is usually mentioned as one of the cardinal features of the disease, but it has hardly proved so in our cases. Naturally it is often with difficulty that lethargy and asthenia can be distinguished from one another, for a lethargic patient will hardly appear to be overflowing with energy. In some of the cases described in the literature the patient is said to have been so weak that he was unable to turn in bed. Many of the present cases attained this immobility in the later stages, but this was due rather to mental lethargy than to muscular weakness.

Tremors. In several instances marked tremors were observed. In one of the first cases of the epidemic there were tremors all over the body which closely resembled the fibrillary twitchings of progressive muscular atrophy. In other cases there was what appeared to be a general trembling of the muscles which could best be appreciated when a hand was laid on the patient. One case displayed extraordinary clonic contractions of the rectus abdominis at the rate of about twenty to the minute and lasting for more than two days.

Expression. In the later stages the face assumes an appearance of gravity and placidity which resembles that of a waxen image. This may be due to two distinct causes. In many cases it is doubtless due to lack of emotional tone, but in others the cause of the immobility is bilateral weakness of the facial nerves. One case of double seventh nerve paralysis presented a face as unchangeable and inscrutable as that of the sphinx, although the patient was by no means dull emotionally. In many cases the Parkinsonian facies so characteristic of paralysis agitans and lesions of the corpus striatum and globus pallidus may be recognized.

Sensation. Disturbance of sensation is not a common symptom, but we have had four instances of what may be called severe neuralgic pains in the early stages of typical examples of the disease. In each of these cases there was severe burning pain in the fingers, hand and forearm, lasting for from one to two days, and unaccompanied by any sign of inflammation. There was no pain either on movement or pressure.

Headache is a fairly constant feature, but in many of our cases has been entirely absent.

Reflexes. When the pathology of the condition comes to be discussed it will be found that the lesions are not such as would lead one to expect any constant alteration in the reflexes, and in practice this proves to be the case. Occasionally, however, the knee jerks may be either abolished or exaggerated, and in several cases there was a positive Babinski on one or both sides. These features are due probably to involvement of the pyramidal fibres as they pass down through the internal capsule and mesencephalon.

Paralysis of extremities. In only one case was weakness of the limbs noted. This occurred in a man in whom the symptoms were almost entirely cortical, although associated with well-marked lethargy. After a series of five or six severe Jacksonian attacks in the left arm and leg he was left for several days with quite distinct weakness on that side. In some of the English cases atrophy of groups of muscles followed the disease.

Mental state. After having seen a number of cases, the observer becomes able to detect a certain mental condition which might be described as characteristic, although it is by no means present in every case. Lethargy is, of course, the most prominent feature, but it is not the lethargy of the comatose or the dope fiend. The patient appears to be plunged in a brown study, his thoughts would seem to have been laid away on the dusty shelves of forgetfulness, and he himself to have entered on a state of hibernation. And yet in many cases the intellect is wonderfully clear.

When the patient is aroused by being spoken sharply to, he may reply with an acuity which sometimes startles the physician. In many cases the condition appears to be one of paralysis of emotional tone rather than of ideation, although in the later stages all spheres of mental activity are equally involved. This possible dissociation of emotional from ideational disturbance is of interest in connection with the similar condition in the progressive lenticular degeneration of Wilson's disease, and also with the well-known dissociation in dementia præcox. Katatonia and other phenomena met with in dementia præcox have been observed in several of our cases.*

Cerebral symptoms. The common occurrence of headache has already been referred to. In some cases it was frontal, in some occipital. Vertigo and giddiness were present in a few cases. No definite examples of cerebellar ataxia, described by other writers, were observed. When it does occur it is probably due to interference with the connections of the superior cerebellar peduncle with the red nucleus in the mid-brain.

Tinnitus was present in so many of the cases early in the disease that it came to be regarded as a symptom of great diagnostic importance.

Diplopia, interference with accommodation, and disturbance of vision were amongst the commonest symptoms, and photophobia was present in one instance. A most characteristic feature was the frequently fleeting nature of these disturbances. They would be here to-day, gone to-morrow, and indeed they varied much from hour to hour. In this respect they are in marked contrast to the symptoms and lesions of poliomyelitis.

Cranial nerve disturbances. The neurologist may divide cases of lethargic encephalitis into three main groups, depending on whether the chief lesions are in the mid-brain, the pons and medulla, or the cerebral cortex. The site of election of the disease is the mesencephalon, and especially the peri-aqueductal group of nuclei which govern ocular movement. It is natural, therefore, that ophthalmoplegia, both internal and external, with the associated symptoms of diplopia, interference with accommodation, disturbances of vision, giddiness, nystagmus, and ptosis should be of frequent occurrence. These were the most characteristic features in our cases, as they have been wherever the disease has broken out. Equally characteristic, however, was the fleeting nature of the disturbances. The nerves involved were the third, probably the fourth, and in one or two cases the sixth. One case of paralysis of

*Dr. Hunter has drawn attention to the slowly fading smile seen in some cases, this being one manifestation of katatomia.

the sixth nerve when seen a few days later showed bilateral paralysis of the seventh nerve, but the sixth nerve palsy had completely disappeared. The pupillary reactions were usually sluggish, but in few cases were they completely lost.

Involvement of the lower group of cranial nerve nuclei in the lower part of the pons and the upper part of the medulla, in those, namely which were in relation to the floor of the fourth ventricle, was frequently observed. Reference has already been made to a case showing bilateral paralysis of the seventh nuclei. In another case which occurred at the beginning of the epidemic the patient presented what appeared to be a typical Bell's paralysis involving the whole of the left side of the face. There was a history of transient diplopia and tinnitus, but stress was not laid on these. It was not until the temperature rose and the patient began to pass into a state of lethargy that a suspicion was aroused as to the true nature of the condition. Slight weakness of the face on one or both sides could be detected in many cases which never developed any pronounced facial paralysis.

Disturbances of the eighth nerve (chiefly tinnitus) and the twelfth nerve have been not infrequent, but none of the other nerves in the pons and medulla have been involved. It is, of course, difficult to test such a nerve as the fifth owing to the lethargy of the patient. Optic neuritis has not been observed in the few cases examined.

Hiccough. Reference may here be made to the subject of hiccough. A few days after the first cases of lethargic encephalitis had begun to appear, a remarkable number of cases of persistent and aggravated hiccough were reported by the Winnipeg practitioners. Several doctors developed the condition, one of whom kept hiccoughing at intervals of a minute or less for five days. In most instances the attack lasted for about forty-eight hours. Over fifty cases of this severe form of hiccough have occurred in the city, and it is probable that there have been many more cases which have not been reported. A few of the cases have been accompanied by slight degrees of fever. There may be no connection between the two epidemics, but it may be noted that two of the cases of encephalitis displayed this symptom at the beginning of the illness.

Course and prognosis. It will be seen from the histories of some of the cases given later that the course of the disease varied as much as the clinical picture. Some of the cases could be described as fulminating. Thus in No. 6 the little patient was dead forty-eight hours after the appearance of the first symptoms.

Other fatal cases lasted not more than three or four days. The usual duration of the fatal cases was a little over a week. In the cases which recovered, the course was variable. The stupor in the milder cases cleared up after a few days, although a certain amount of lassitude might remain for a considerable time. In many instances the illness dragged on for a number of weeks, the patient being better one day and worse the next.

The temperature curve was of little assistance in prognosis; it certainly did not run parallel with the clinical condition of the patient. None of the cranial nerve palsies were of very great duration, although in some of the English cases they lasted for three months. On the whole the prognosis is better than the alarming appearance of the patient at the height of the illness would seem to indicate. In several cases a fatal outcome appeared certain, yet the patient recovered. In the English epidemic thirty-seven deaths occurred in one hundred and sixty-eight cases, a mortality of 22 per cent. Netter reports seven deaths in fifteen cases in the Paris outbreak, and Economo five out of eleven. In the Winnipeg epidemic, twenty-three cases out of sixty died of the disease, a mortality of 38 per cent.

Blood. Leucocyte counts were made on all of the cases which came into hospital. In a few there was a moderate degree of leucocytosis, the highest being 16,000, but in the great majority the count was quite normal, even when a considerable degree of fever was present. The differential count showed no variation from the normal.

Cerebrospinal fluid. The accounts of the condition of the spinal fluid contained in the literature vary considerably. English observers found little or no change in the greater number of cases, whereas more constant positive changes were found in the American cases. In some of our cases the fluid was normal, in others there was a moderate degree of lymphocytosis, and in two cases the increase was marked, 154 cells in the one case, 210 in the other. A probable explanation is that the condition of the fluid varies from day to day. Wegeforth and Ayer found variations in the same fluid at different times, and one of our cases showed four cells on one occasion and thirty-eight cells three days later, although by that time the condition of the patient was better rather than worse. Differential counts showed that the cells were practically all lymphocytes. The condition of the spinal fluid appears to bear no relation to the severity of the illness. Both of our cases with high counts recovered, and in most of the fatal cases the fluid was normal. The globulin was either normal or very slightly increased, even in

the cases in which the cell count was high. The Fehling-reducing power was normal. The condition of the fluid of course merely indicates whether or not the meninges are involved in the inflammatory process. An inflammation deep down in the basal ganglia or in the centre of the mesencephalon may leave the meninges untouched and produce no change in the spinal fluid.

Nature of the Disease

The cause of epidemic encephalitis, although certainly microbic in nature, is still unknown. Nevertheless it may be possible to arrive at a conception of the nature of the condition from analogy and from what may be termed circumstantial evidence. Three main views are held at the present moment: (1) that it bears a close relation to, and is indeed a complication of, influenza; (2) that it is a cerebral form of poliomyelitis; (3) that it is a disease *sui generis*.

1. *The influenza hypothesis.* The champions of this theory are able to marshal an array of facts in support of their contention which at first sight are very convincing. They use first the historical argument. Previous outbreaks of what has been termed sleeping sickness have usually followed or accompanied epidemics of influenza. The outbreak of sleeping sickness which occurred at Tübingen in 1712 was associated with an influenzal epidemic at the same time. The mysterious condition named nona, characterized by profound lethargy and drowsiness, followed immediately upon the great epidemic of influenza in 1890. Henoch described a case of polioencephalitis with paralysis of one arm in a girl coming on two weeks after an attack of influenza, and a number of similar cases were described about the same time. The recent outbreaks in Austria, France, England, and America were all associated more or less closely with the influenza epidemic. Further, many cases of influenza are marked by great lethargy and drowsiness, and some cases are said to have slept for several days.

These specious arguments fall to the ground, however, on closer inspection. Darwin's warning that analogy is a deceitful guide is as necessary now as on the day on which he uttered it. To say that the present outbreaks of epidemic encephalitis have followed the influenza epidemic has about as much value from the point of view of proof as to say that they followed the Great War. In a recent paper Heiman goes so far as to call the condition post-influenzal encephalitis, although in one of his eight cases four

months had elapsed since the attack of influenza. The prolonged sleep occasionally associated with influenza is very different from the condition of encephalitis lethargica. In none of these cases have the characteristic lesions in the brain been demonstrated. Finally, the Winnipeg experience seems to prove conclusively that no ætiological relationship exists between influenza and epidemic encephalitis. The present outbreak is typical in every respect, including the microscopic findings in the brain, but it is a year since influenza visited the city, and not one of the cases had had a recent attack. Indeed, in a majority of the cases there was no history at all of influenza.

2. *The poliomyelitis theory.* The points of resemblance between poliomyelitis and lethargic encephalitis are many and close. As the grey matter of the anterior horns of the spinal cord is traced upwards into the brain stem it becomes broken up by the decussations of the pyramid and fillet, so that many of the cranial nuclei, more especially those of the sensory and mixed nerves, come to assume a lateral position. The purely motor nuclei of the third, fourth and sixth nerves, however, maintain their median relation. Poliomyelitis is a disease which in its typical form attacks the grey matter of the anterior horns of the cord, whilst in lethargic encephalitis the brunt of the attack falls on the grey matter of the brain stem, especially the nuclei of the third, fourth, and sixth nerves. Both diseases are acute in onset, febrile in nature, motor in their manifestations, although in both there may be disturbances of sensation.

A closer scrutiny reveals points of similarity of even greater importance. Poliomyelitis can no longer be regarded as a disease involving only the spinal cord. Just as cerebrospinal fever is a systemic infection usually attacking the central nervous system, but in some cases sparing that system, so poliomyelitis must be regarded as a general infection in which other organs than the cord may be involved. As Peabody, Draper, and Dochez have shown in their masterly monograph, there is widespread involvement of the lymphoid apparatus, of the liver and other parenchymatous organs, and of the brain. The same lesions which are so characteristically found in the cord, and which are so similar to those of lethargic encephalitis, are also met with in the pons and medulla. Although not hitherto so described, there can be no doubt that lethargic encephalitis is also a systemic infection, as will be seen in the discussion of our pathological findings, with the production in most cases of characteristic lesions in the brain. The fact that

very similar changes may be found in the brain in both diseases is a fact which cannot be ignored. The essential changes are perivascular infiltration, diffuse inflammatory infiltration, hæmorrhages, and destruction of nerve cells. All these are found in both conditions, but there are certain points of difference, possibly fundamental in character, which will be discussed under the heading of pathology.

On the clinical side the resemblance between poliomyelitis and lethargic encephalitis is equally striking. Since the days of Wickman an encephalitic form of poliomyelitis has been well recognized. Peabody, Draper, and Dochez describe a number of cases of most evident inflammation of the grey matter of the brain. This may be associated with spinal paralysis, or may occur alone. Facial paralysis occurred in eight out of twelve cases of combined cord and bulb lesions, and ocular palsies were present in four cases. Nystagmus, diplopia, strabismus, and disturbances of speech were all met with. Four of Peabody's cases, moreover, displayed a marked condition of drowsiness deepening into stupor. The patients lay in a sort of coma vigil, and the face assumed a peculiar mask-like immobility. The temperature was elevated. The stupor, after lasting several days, cleared up with remarkable rapidity in the course of a few hours. In a series of four hundred cases Batten found that 12 per cent. showed evidence of encephalitis involving the medulla, pons, or mid-brain.

It must be admitted, then, that there are many striking points of similarity between poliomyelitis and lethargic encephalitis, both as regards clinical manifestations and pathological lesions. This does not mean, however, that they are necessarily one and the same disease. Diverse lesions in the brain may produce a very similar clinical picture. Both tuberculous meningitis and cerebral tumour may be the cause of lethargy and drowsiness, headache, diplopia, and cranial nerve palsies. Further, in different diseases the pathological picture may be remarkably similar. The fact that perivascular infiltration and degeneration of ganglion cells are found both in poliomyelitis and encephalitis lethargica does not prove that they are due to the same cause, for practically identical changes are found in cerebral syphilis and in trypanosomiasis, diseases of which the cause is definitely known.

If the arguments for the poliomyelitis hypothesis are strong, those against are even more formidable.

1. Epidemics of poliomyelitis occur with remarkable constancy in the summer time; the outbreaks of encephalitis have occurred during the winter months.

2. Poliomyelitis is a disease, par excellence, of children; encephalitis is much more common among adults than children.

3. The onset of paralysis in poliomyelitis is typically sudden, the effects are lasting, and there is usually muscular atrophy; in encephalitis the palsies often come on gradually, are characteristically fleeting, and there is no muscular atrophy.

4. If the two diseases are due to the same cause, it is strange that in the present epidemic no cases of spinal poliomyelitis should have occurred.

5. The virus of poliomyelitis is readily transmitted to monkeys, whereas no cases of satisfactory and undoubted transference have been reported in encephalitis.

6. Although the virus of poliomyelitis is introduced intracerebrally in monkeys, the lesions produced are always spinal, never cerebral.

7. Although many cases of poliomyelitis may show lethargy, even coma, yet with the onset of respiratory difficulty, as Peabody has pointed out, the mental state becomes clear, and the child seems to awaken to the struggle that lies before it. Nothing like this is seen in lethargic encephalitis.

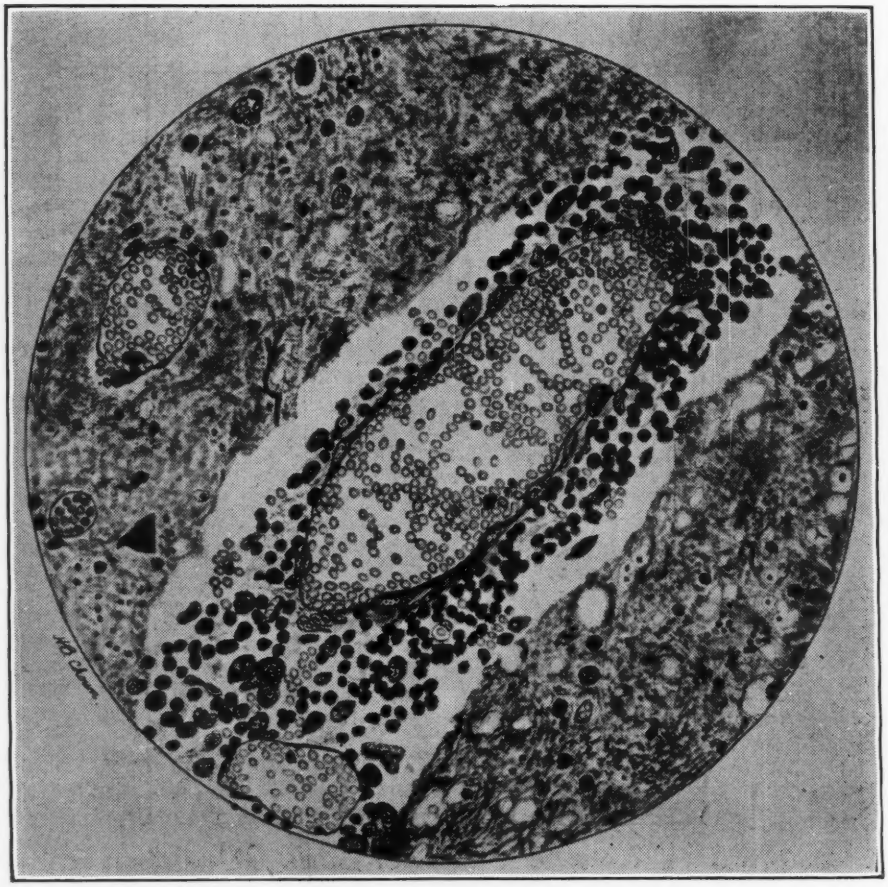
8. Leucocytosis, sometimes as high as 30,000, is met with in poliomyelitis. It is usually normal or only slightly raised in encephalitis.

9. A lymphocytosis, sometimes marked, in the cerebrospinal fluid is the rule in the early stages of poliomyelitis; the count is normal or only slightly increased in encephalitis except in exceptional cases.

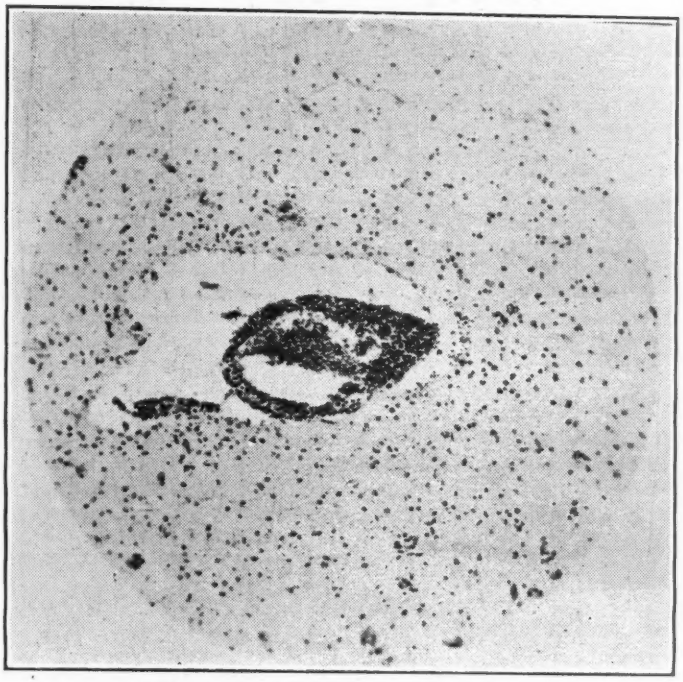
10. An attack of poliomyelitis is supposed to confer practically complete immunity, so that true second attacks, apart from relapses, are almost unknown. Batten refers to three very doubtful cases in the literature, but considers that none of them can be accepted as undoubted examples. In one of our fatal cases the patient had had a typical attack of poliomyelitis in childhood which left him with permanent weakness of one leg.

11. The ultimate decision will rest with the bacteriologist or pathologist. The bacteriological evidence, as stated above, is against the theory so far. The pathological evidence is uncertain. The evident changes in the two conditions are of course similar. Some of our work in the present epidemic, however, seems to suggest important differences.

Showing marked degree of ptosis and slight weakness of the left facial nerve. The patient is trying to look at the camera.



Vessel in mid-brain showing perivascular infiltration with small lymphocytes and larger plasma cells.



Round-celled infiltration around vessel in mid-brain.

CLINICAL CASES

It is not possible, nor indeed desirable, within the limits of this paper, to give details of all the clinical cases. They have accordingly been divided into the following groups, two or three examples of each being given: (1) mesencephalic; (2) subthalamic; (3) bulbar; (4) cortical; (5) apoplectic; (6) septicæmic; (7) maniacal. This is, of course, far from a satisfactory or scientific classification, being partly anatomical and partly based on symptoms. It is rather a series of heads under which may be described the clinical symptoms which presented themselves in the present epidemic.

Mesencephalic Type

1. *Fatal case with ocular and sensory symptoms.* F. E. C., male, aged thirty-seven, admitted to hospital on November 9th, under Dr. Hunter. For two weeks before admission he had complained of general weakness and loss of appetite, and at times felt unduly sleepy. On November 6th he felt so weak that he was unable to go to work. He suffered severe pain in the right shoulder and upper arm for which poultices had to be used, and which lasted for two days. He became very drowsy and lethargic on November 8th, and twitchings were noticed in the arms, face and lips.

On admission the patient was in a profoundly lethargic condition, taking no notice of what was going on and lying motionless in bed with closed eyes and a somewhat flushed face. When spoken to, however, he answered with clearness and accuracy. There was right-sided ptosis, inequality of the pupils, and slight weakness of the right seventh nerve. The pupils reacted but faintly to light and not at all to accommodation; the fundus was normal. Irregular tremors appeared to play over the body, now affecting the face, now the arms and legs. The knee jerks were normal, and there was no Babinski sign. The temperature was 103°, pulse 120, respirations 24. There was a leucocytosis of 16,000, and the cerebrospinal fluid was normal, apart from a slight increase in pressure.

During the next three days the lethargy deepened into coma, the ptosis became bilateral, marked cyanosis developed, and the patient died on November 12th, with a temperature of 104·5°. At no time during his stay in hospital was his temperature below 100°.

At autopsy there was marked congestion of the cerebral vessels, dilatation of capillaries in the mid-brain and the medulla,

but no gross hæmorrhages. Microscopic examination showed characteristic patches of perivascular infiltration and cellular degeneration throughout the brain stem.

2. *Severe case with typical onset.* T. R., male, aged forty-four years, admitted to hospital on November 21st, under Dr. E. W. Montgomery. The patient, a street-railway motorman, was in the best of health until November 19th, when he felt a "soreness" on the left side of the head which he was unable to localize. He felt well otherwise, and worked full hours. On the following morning he did not feel at all well, his legs were so shaky that they almost gave way under him, his head was worse, and he was relieved from duty at 4 p.m. Next day he felt better, got up at 5 a.m., and worked for two hours. His legs then became weak, and he noticed that when a car approached him there appeared to be two cars, one upon the track on which he was driving and one on the other. He went off duty chiefly because of the weakness of the legs, but by that time he was beginning to feel drowsy.

On admission the patient presented a typically febrile appearance, the face was markedly flushed, the eyes were heavy, the tongue furred, the temperature was 100·4° and the pulse 84. He was unable to shut the right eye, the pupils reacted sluggishly to light, but there were no other signs of cranial nerve palsies apart from the diplopia, which was still very pronounced. The cerebrospinal fluid was normal, and the leucocytes 7,200. During the succeeding days drowsiness alternated with periods of great restlessness, and the patient rapidly lost ground, his face acquired a pinched appearance, and although the temperature gradually returned to normal, there can be little doubt that the case will terminate fatally.

3. *Fatal case with marked insomnia.* A man, aged twenty, under the care of Dr. Chestnut, noticed a twitching of the left upper eyelid. Next day a temporary diplopia developed, lasting only twenty-four hours. About the same time he complained of what he described as a soreness in the right upper arm, and later in the left forearm and hand. There were no signs of local inflammation, nor pain on movement or pressure. Hiccough was a troublesome feature during the early stages of the illness. From the first, insomnia was marked. On several occasions there were fleeting noises in the left ear. The patient was nervous and excited, and on the second night of the illness he went to a dance. On the fifth day he began to exhibit drowsiness during the day, although wakeful at night. Distinct ptosis was noticed, and he complained

of feeling "jerky", although there were no definite twitchings of the muscles. He rapidly became very drowsy and lethargic, although restless and sleepless at night, and died in a state of coma on the eighth day of the illness.

Sub-Thalamic Type

4. *Case resembling acute paralysis agitans.* Miss K., aged twenty-one years, seen with Dr. Field on November 24th. Nine days before she began to feel dull and out of sorts, complaining of a buzzing noise in the left ear. She suffered from insomnia, and during the day was alternately apathetic and restless. There were no eye symptoms nor undue drowsiness. The temperature was normal. She presented a most striking picture of Parkinson's disease, sitting leaning forward in her chair, with her head stooping forward as if she had a stiff neck, and the blank, expressionless appearance of a mask. When she walked, the gait was typical to a degree, shuffling, the back bent forwards, the arms flexed at the elbow and the hands at the wrist, the rigid gaze fixed on the ground. There were irregular tremors of the arms and legs, the knee jerks were increased, there was no paralysis of the cranial nerves, the pupils reacted well to light and accommodation. In such a case as this the lesions must almost certainly be in the same region as is involved in paralysis agitans, namely the putamen and globus pallidus.

Bulbar Type

5. *Mild case with mainly local symptoms.* A. M., a man aged thirty-five, seen with Dr. Victor Williams. About October 25th, he began to suffer from violent throbbing in the head and distressing dreams at night. These were succeeded by pain in the suboccipital region, vomiting, slight diplopia, and noises in the left ear. When seen on November 1st, there was complete facial paralysis on the left side and very slight lateral nystagmus. The pupils reacted normally, and there was no other sign of an oculomotor lesion. The patient was rather lethargic, but the temperature was normal. As this case occurred before the beginning of the outbreak of encephalitis, a diagnosis of ordinary facial paralysis or possibly of tumour in the cerebello-pontine angle was made. A few days later, however, the patient became extremely drowsy, and the correct diagnosis was then evident. At the end of ten days the drowsiness had completely cleared up, as had the facial paralysis, except for slight weakness in the lower part of the face.

Cortical Type

6. *Fatal case with cortical symptoms.* A girl seven years of age, admitted to hospital under Dr. Field on November 12th. She was taken ill suddenly on the morning of the previous day with headache, vomiting, ringing in the ears, failure of vision, unsteadiness in walking, and convulsions, of which she had six during the day. No diplopia nor strabismus. During the time that she was in hospital she had a series of attacks of typical Jacksonian epilepsy, in which there were severe convulsions of the right arm and leg, and the head was jerked over to the right side. Towards the end of the seizure the spasms extended to the left leg. The attacks lasted from five to ten minutes, and during them the patient was quite conscious. In the intervals she lay quietly in bed, but showed no trace of drowsiness. There was albumen in the urine, together with epithelial cells and red blood corpuscles, so that a diagnosis of uræmia was considered. She died early on the morning of November 13th. The temperature at no time was above 102°, and shortly before death it went down to 96°.

The autopsy showed marked flattening and œdema of the convolutions in the left frontal region, and the characteristic microscopic lesions of encephalitis were found in the brain stem. There was extreme congestion of the kidney, hæmorrhage into the collecting tubules, and degeneration of the convoluted tubules.

7. *Mild case with cortical symptoms.* J. H., male, aged thirty-two years, was admitted to hospital on November 13th, under Dr. Moody. On the morning of that day he noticed a noise as of whistling in both ears, and suffered from a slight headache. In the afternoon he visited a friend in the hospital, went down town feeling much better, entered a store, and then was suddenly overcome by weakness; the room appeared to go round him, and he would have fallen had it not been for the assistance of a companion. He experienced great weakness in both legs, more especially the right, and also in the right hand. He was taken back to hospital, and on admission it was noticed that there was rigidity and twitching of the right leg. An hour later he had a typical Jacksonian attack involving the right arm and leg, but not the head. There was a positive Babinski sign on the right side. He now became very drowsy, and slept for the greater part of the succeeding three days. He had five more convulsive attacks, but none after the second day. The temperature was 101° on admission, and remained

elevated for five or six days. The leucocytes were 7,600, and the cerebrospinal fluid was normal, except for a moderate increase in pressure. The patient made a good though gradual recovery. The temperature gradually returned to normal, the drowsiness departed, but there was weakness of the right arm and leg and a positive Babinski on the right side for fully a week.

Apoplectic Type

8. *Sudden onset with aphasia.* Mrs. J., aged fifty years, was admitted to hospital on November 23rd. She was perfectly well on the morning of that day. At 5 p.m. she suddenly staggered to a wall, and would have fallen but for her husband. She was laid on a couch in what was described as an unconscious condition. She was unable to speak. Spasmodic movements of the left arm and leg were noticed. On admission four hours later, she was extremely drowsy but not unconscious, the left arm and leg were stiff, the deep reflexes in those limbs were exaggerated, ankle clonus and a positive Babinski sign were present on the left side, the mouth was drawn slightly to the left. The temperature was 96°, pulse 70, respiration 26. Red blood cells were present in the urine and a large amount of albumen and hyaline casts. There was much doubt at first as to whether the case was one of ordinary apoplexy or lethargic encephalitis.

For two days she lay like a log, very drowsy, with eyes closed, uttering no word, making occasional movements with the left arm and leg. She has gradually improved and is now quite bright mentally, but is still unable to speak. She has regained the use of her limbs. The urine is now normal.

9. *Apoplectic onset with aphasia and hemiplegia.* A girl of ten years went to bed feeling perfectly well. Two hours later she awakened with a cry, and her parents found her tossing about the left arm and leg, but the right side remained motionless. She was unable to speak. When seen by Dr. E. W. Montgomery she was found to show a right-sided hemiplegia of upper neurone type with a positive Babinski and paralysis of the internal rectus on the left side. She was completely aphasic and extremely drowsy.

10. *Fatal case resembling apoplexy.* M. V., a man aged forty, after an indefinite history of headache, suddenly passed into a state of coma from which he could not be aroused. He lay on his back breathing stertorously, the right arm and leg were spastic, and there was incontinence of urine. The clinical picture was so

strongly suggestive of cerebral hæmorrhage that that diagnosis was made. He died forty-eight hours later, and at the autopsy the brain presented a remarkable appearance. The whole of the left cerebral hemisphere was studded with petechial hæmorrhages, more marked in the white than in the grey matter, and the brain substance was of a distinct pink colour. There were a few lesions of a similar nature in the right hemisphere. The basal ganglia, mid-brain, pons and medulla were all extremely congested, the change being most marked in the substantia nigra. Microscopic examination showed numerous hæmorrhages and other signs of acute encephalitis throughout the brain, marked congestion and degeneration in the kidney, and a lesser degree of congestion in the liver.

Septicæmic Type

11. Mrs. M., aged forty-two years. Ill for seven days with headache, diplopia, ptosis, strabismus, and marked drowsiness and lethargy.

Post-mortem examination by Dr. Bell showed widespread petechial hæmorrhages over the pleura, pericardium, and diaphragm, and blood-stained effusion in pleural and peritoneal cavities. The kidneys were very soft and flabby. Microscopic sections showed most remarkable changes which are described more fully under the heading of pathology. Not only was there extreme congestion of both cortex and medulla, and profound degeneration of the convoluted tubules such as might be found in corrosive sublimate poisoning, but there were focal collections of round cells in the boundary zone strongly suggesting scattered foci of infection. The pial vessels were extremely congested, and there was marked congestion of the mid-brain, notably in the substantia nigra. The microscopic appearances in the mid-brain were of the usual character.

12. G. P., aged thirty-eight years, developed headache and diplopia, and soon became very drowsy. The lethargy deepened with remarkable rapidity, although he could be aroused almost until the end. No cranial nerve palsies were noted. The illness lasted only two days, and the patient died in a state of coma. There was moderate fever throughout the illness. It is important to note that the patient had an attack of infantile paralysis when a child, which left him with a typically weak and atrophic leg.

The post-mortem examination by Dr. Bell revealed widespread hæmorrhages over the pleura, pericardium and diaphragm. The

pial vessels were very congested, and the substance of the cerebrum was much softer than normal. There was considerable congestion of the mid-brain, and the usual microscopic changes were found.

13. T. D., aged fifty years. For several days he had been ill with typical symptoms of lethargic encephalitis, and was found dead in bed. At post mortem the parietal and visceral pleura, pericardium, and diaphragm were covered with petechial hæmorrhages. The body was so decomposed, owing to having been kept for several days in a warm room, that microscopic examination of the organs was impossible.

Maniacal Type

14. A man fifty years of age, living in the country, on getting up one morning began to pray. This struck his wife as being unusual, for it was not the custom with him. He continued at his prayers all morning, and later in the day became very excited. He persisted in crawling under the bed, and had to be strapped down to the mattress. There was no trace of drowsiness or lethargy. He remained acutely maniacal for several days, and was then sent by train to the Psychopathic Hospital in Winnipeg, but died in the ambulance.

At the post mortem, hæmorrhages were found in the floor of the fourth ventricle, and microscopic examination revealed the characteristic lesions of lethargic encephalitis in the medulla, pons and mid-brain.

Pathology

Encephalitis lethargica must still be classed among the diseases of mystery. Its cause is unknown, and the pathological changes found in the brain do not throw conclusive light upon the nature of the condition. Nevertheless, quite definite microscopic changes are found, changes from which alone it is sometimes possible to make a final diagnosis in the more obscure cases. These changes cannot at present be described as being pathognomonic. This, however, is hardly to be wondered at, for the lesions in brain diseases are limited in variety, and several very different clinical entities present not dissimilar microscopic appearances.

Post-mortem examination was made in eighteen cases either by Dr. Bell or myself. Permission was obtained to remove the kidney in two private cases in which it was not possible to perform a com-

plete autopsy. The task of examining this extensive material by modern neurological methods is a matter of time, and a more detailed report of further researches on the brains and kidneys will be made in a subsequent paper. All that is at present attempted is to present the results of a preliminary and necessarily incomplete investigation.

Brain. The brain may appear perfectly normal to the naked eye, although marked changes may be found on microscopic examination. In many cases a varying degree of hyperæmia was observed in the brain-stem; being most marked as a rule in the region of the substantia nigra in the mid-brain. The substantia nigra was less clearly defined than in the normal brain, and in some cases could be distinguished with difficulty. In one case (No. 10) there were petechial hæmorrhages throughout the whole of one cerebral hemisphere, and congestion so great that the brain substance was of a distinct pink colour. Three cases showed small hæmorrhages in the floor of the fourth ventricle.

Microscopically the outstanding features have been capillary congestion and perivascular infiltration. These changes have not been found by any means uniformly throughout the brain. In the cortex, congestion has been present, but seldom any degree of perivascular change. The most pronounced changes have been in the mid-brain and medulla, and to a lesser extent in the pons and basal ganglia.

The perivascular changes are the most striking and interesting. The affected vessel is separated from the brain substance by a clear space varying in width, in some cases containing numerous red cells which have escaped from the vessel. This appearance is perhaps due to an inflammatory œdema. The vascular endothelium is unusually distinct, owing to swelling of the cells which may come to resemble fibroblasts. The wall of the vessel is infiltrated with a collar of cells which is of fairly uniform thickness around the vessel. Some of these cells are lymphocytes, but the majority in our sections are unmistakably plasma cells, with a much larger and more vesicular nucleus, often placed eccentrically, and abundant cytoplasm, the outline of the cell being often polygonal rather than round. No evidence of invasion of the surrounding brain matter by these cells could be found, so that the diffuse infiltration of other writers was not corroborated. A study of the sections gave the impression that the brunt of the attack had fallen upon the vessels, and that any changes in the nerve elements would probably be secondary. Distinct hæmorrhages were present

in some sections, but they were by no means a prominent feature, except in one or two cases where they were evident to the naked eye.

The nerve cells showed in places changes of great interest, but these are still under investigation. Varying degrees of cellular dissolution were present, from slight chromatolysis to complete disappearance of the Nissl granules and even of the cell body. In some of the cells there was an abundant yellow granular pigment, lighter in colour than that normally present in the substantia nigra, and resembling the pigment found in the senile brain. In places the body of the cell had disappeared, its position being indicated by this yellow pigment. Considering that the fatal cases were of only a few days' duration, it is indeed remarkable that such profound destruction coupled with so much pigmentation should have occurred. A detailed examination of the cranial nerve nuclei in the mid-brain, pons and medulla has not yet been made.

Some definite relation between the vascular and cellular changes would be expected, whether the cellular degeneration was secondary to the vascular changes or was the result of the action of a virus situated in the blood vessels. No such relation, however, could be found. The most inflamed vessels might be surrounded by apparently normal nerve cells, and in places where the cell bodies were mere shadows there might be no vascular change. The pathology, then, may be regarded as an acute interstitial inflammation with parenchymatous degeneration, no clear connection being established between the two conditions.

Meningeal involvement was slight and variable. In many cases none could be found, in others there was a moderate degree of infiltration around the vessels. The changes in the spinal fluid are probably directly dependent on those in the meninges.

Kidney. An observation of great importance is that other organs than the brain may show marked changes. It is rather remarkable that in the records in the literature attention appears to have been focussed on the brain so exclusively that no microscopic examination of other organs was made. This was unfortunately also true of our own earlier cases, but it was soon realized that a more general examination was necessary. It was found that striking lesions were present in the kidneys in all of eight cases examined. There were two chief types of change, extreme congestion of the vessels, most marked in the medulla but also present in the cortex, and great degeneration of the convoluted tubules. Not only were the medullary vessels distended to such a degree as to obscure the tubules, but definite hæmorrhage into the tubules

had occurred in many places. The vascularity was so great as to recall at times the edge of an infarct. The parenchymatous degeneration varied in degree, but in one case it amounted to complete necrosis of the cells lining the tubules, the picture suggesting the action of some powerful irritant on the renal cortex. The glomeruli showed considerable congestion and in some there was slight hæmorrhage into the capsular space; there was no change in the capsular epithelium. The contrast between the convoluted and collecting tubules was most marked; a convoluted tubule would show extreme disintegration whilst an adjoining collecting tubule would be apparently normal. The cells lining the ascending loop of Henle showed a similar change, although less marked in degree. The brunt of the attack, then would seem to fall on the vessels and the filtering apparatus of the kidney.

The collecting tubules, however, did not always escape so lightly as the above description might imply. Here and there in some cases the cells lining these tubules were crowded with fine yellow granules. The exact nature of these granules has not yet been determined. They were most numerous in the case showing widespread hæmorrhages throughout the cerebrum, and it is probable that they consist of blood pigment. In this case the walls of some of the collecting tubules stained so darkly with iodine as to suggest an amyloid reaction, a slighter degree of the change being also noted in the glomeruli. In one case there were abundant granules in the collecting tubules giving a fat reaction with Scharlach R in frozen sections, although the convoluted tubules which were profoundly disintegrated showed no trace of fat. This remarkable finding may have been due to the processs having been so acute in the convoluted tubules that there was no time for the fatty change to take place, whereas the slighter lesions in the collecting tubules allowed time for the reaction to occur.

In one case (No. 11) an additional lesion was noted. In the boundary zone between the cortex and medulla there were foci of round celled infiltration strongly suggesting the presence of a bacterial as opposed to a toxic irritant. These were quite localized, and it was not possible to determine whether they bore any definite relation to the vessels. The patient had been ill for seven days. At the autopsy there were widespread petechial hæmorrhages, and blood-stained serous effusion in the pleural and peritoneal cavities.

Urinary changes would be expected in view of such a condition of the kidney. Blood and albumen were present in several of the cases, one of which at autopsy showed the usual lesions in the

kidney. Some of the kidneys were obtained from outside cases, in which there was no record of the condition of the urine.

Observations on other parenchymatous organs are being made, but the results are not ready for publication.

It would appear from the above observations that there is sufficient evidence to show that lethargic encephalitis is not merely an inflammatory condition of the brain, but a general infection involving many parenchymatous tissues in which the brain is the chief sufferer. This conception falls into line with modern views regarding other infective conditions of the central nervous system. General paralysis, trypanosomiasis, cerebrospinal meningitis, and acute poliomyelitis are now regarded as general infections with special localization in the brain and cord. In view of the many points of similarity between lethargic encephzlitis and acute poliomyelitis it is of interest to note that Peabody and his co-workers found constant lesions in the liver and the lymphoid tissues, especially in Peyers patches.

All bacteriological investigations have proved negative. Cultures of the blood and the cerebrospinal fluid gave no result. Dr. Bell and Dr. Cadham emulsified portions of the brain and injected it into the brain of rabbits, but without effect. Dr. Nicholson did the same with the cerebrospinal fluid with a similar result.

Summary

1. The Winnipeg epidemic of sixty cases with twenty-three deaths corresponded closely with previous epidemics already described. The mortality of 38 per cent. was unduly high.

2. The characteristic case presenting fever, drowsiness, strabimus, ptosis, diplopia, tinnitus, some degree of facial weakness, constipation and perhaps some urinary and spinal fluid changes, is readily recognized. The fleeting nature of the disturbances is very typical. Sensory disturbances were present in a number of cases. Some of the cases were atypical, suggesting cerebral tumour, apoplexy, and other brain lesions.

3. The brain was examined in eighteen cases, and showed marked congestion, perivascular infiltration with lymphocytes and plasma cells, and occasionally hæmorrhage. Degeneration of the nerve cells was variable. The changes were most marked in the mid-brain. Marked lesions were also found in the kidneys.

4. A remarkable epidemic of hiccough occurred in this city at the same time as the outbreak of encephalitis.

In conclusion I wish to express my great indebtedness to the numerous physicians who have so generously placed their clinical material at my disposal, to Dr. Gordon Bell for providing me with much post-mortem material and for valuable assistance in solving some of the pathological problems which presented themselves, and to Miss M. van Romburgh for the devotion and care with which she has prepared the microscopic material.

BIBLIOGRAPHY

Von Economo.—*Wien. Klin. Wchnsch.*, 1917, 30, 581.

"Report of an Enquiry into an Obscure Disease, Encephalitis Lethargica." Local Government Board, N.S. 121.

Alexander, J. B.—"Cases resembling Encephalitis Lethargica occurring during the Influenza Epidemic, *B. M. J.*, 1919, i, 794.

Bassoe, P.—"Epidemic Encephalitis," *J. A. M. A.*, 1919, 72, 971.

Claisse, P.—"Encéphalite Léthargique." *Bull. et mém. Soc. med. d. hop. de Paris*, 1919, 43, 222.

Kennedy, F.—"Epidemic Encephalitis with Stupor." *Med. Rec.*, 1919, 95, 631.

Netter, A.—"Recrudescence d'encéphalite léthargique." *Bull. et mém. Soc. med. d. hop. de Paris*, 1919, 43, 300.

Weber, F. P.—"A case of Lethargic Encephalitis with abscess formation in the Brain." *Clin. Journal*, 1919, *48*, 13.

Wilson, S. A. K.—"Epidemic Encephalitis," *Lancet*, 1918, 195, 7.

Heiman, H.—"Postinfluenzal Encephalitis," *Am. J. Dis. Child.*, 1919, 18, 83.

"Discussion on Epidemic Encephalitis," *Proc. Roy. Soc. Med.*, 1918, 12, No. 1.

Buzzard, E. F.—"Encephalitis Lethargica," *Proc. Roy. Soc. Med.*, 1919, 12, No. 9.

Neal, Josephine B.—"Meningeal Conditions noted during the Epidemic of Influenza." *J. A. M. A.*, 1919, 73, 714.

Neal, Josephine B.—"Lethargic Encephalitis," *Arch. Neurol. and Psych.*, 1919, 2, 271.

Wegeforth, P., and Ayer, J. B.—"Encephalitis Lethargica," *J. A. M. A.*, 1919, 73, 5.

Bassoe, P., and Hassin, G. B.—"A Contribution to the Histology and Pathology of Epidemic ("lethargic") Encephalitis," *Arch. Neurol. and Psych.*, 1919, 2, 24.

Peabody, F. W., Draper, G., and Dochez, A. R.—"A Clinical Study of Acute Poliomyelitis," Monographs of the Rockefeller Institute, No. 4.

Batten, F. E.—"Acute Poliomyelitis," *Brain*, 1916, 39, 115.

Lehermitte, Jean.—"L'encéphalite Léthargique," *Ann. de Méd.*, 1919, 6, 306.

OBSTETRIC PARALYSIS—ITS CAUSE AND TREATMENT

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OBSTETRIC paralysis, a paralysis produced during birth, is due to an injury of greater or less extent to the nerves of the brachial plexus. The resultant paralysis is characteristic; the whole arm hangs vertically, the elbow extended, the forearm pronated, and the whole arm inwardly rotated. The paralysis is a flaccid one.

Obstetric paralysis was first described by Smellie[1] in 1768, who believed the condition due to long pressure on the arm while the child was in the pelvis; but it was first brought prominently to the notice of the medical profession in 1872 by Duchenne, who described four cases in infants and attributed the condition to pressure of forceps or fingers in the axilla on the nerve trunks.

Duchenne recognized that the lesion might occur in obstetric operations, such as disengaging the upraised arm in a breech or footling presentation, in delivering after version, or in making traction on the arm of the child after the birth of the head, and quotes cases to support this theory. These procedures result in direct traction on the cords of the plexus, and when force is used probably cause injuries to the nerves. It was not until 1874 that Erb described the same type of paralysis in adults, since which time it has been commonly known as the Erb-Duchenne paralysis. Erb showed that pressure above the shoulder on the junction of the fifth and sixth cervical nerve roots, the so-called Erb's point, caused the characteristic grouping of the paralyzed muscles. He laid the occurrence of the paralysis especially "to the energetic application of the so-called Prague grip in which the fingers are applied like a fork over the back of the child's neck, with an after-coming head, and so endangering the integrity of the brachial plexus by energetic traction and compression".

Stransky[1], in a most careful review of the whole literature up

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to 1902, presents the subject in detail and most conclusively. He reviews Smellie (1768), Danyau (1851), Guéniot (1867), and Depauls' work, the latter cited by Seeligmuller. He reports ninety-four cases from various authors whose works he has reviewed. Stransky believed that pressure as well as hard pulling in some cases was an adequate cause, especially if ether had been used and the child was asphyxiated. The following authors are quoted from Stransky's article:

Seeligmuller thought that pressure from forceps often caused hæmorrhage about the plexus. Thorborn (1886) reported a case of lower arm paralysis, and believed the tearing of the nerves to be due to hyperextension of the shoulder as the arm was drawn above the head, but also ascribed it to pressure of the clavicle on Erb's point from the bad position of the arm.

Roulland (1884) gave all the various positions in which the condition could occur, and apparently believed it due to direct or indirect pressure on the plexus. Arens (1889) believed it due to hæmorrhage or tearing of the nerves.

Kustner (1888) advanced a theory that has been rejected at once by all other writers who have had any extensive experience with the cases, namely, that the trouble is usually due to a fracture of bones or separation of the humeral epiphysis.

Danchez (1891) believed the condition to be spontaneous, from pressure on the circumflex nerve of the arm while the child was caught in the pelvis, or that it might be traumatic from finger or instrumental pressure. He also believed that when the lower arm was involved, the condition was one of the "pseudoparalysis", as also did D'Astros (1892), that is, not a paralysis from nerve injury, but an arm held motionless on account of bruising and consequent pain, or as the result of bone injury. Gowers believed the paralysis to be due to pressure from forceps, and Weil (1896) that it was due to trauma, especially with an after-coming head. Peter thought it due to pressure of the forceps or strong lateral bending of the head, with a delayed shoulder, or turning of the head in breech cases. Guillemot (1896) likewise supported the theory that the condition was due to compression of forceps or a strong pull; and Jolly (1896) believed it due to pressure, chiefly with an after-coming head.

Stransky quotes the experimental work of Fieux (1896), Schoemaker (1899), Stolper (1901), Kustner (1888), and Landold, as follows:

Fieux opposed Erb's views, in that Erb's point was too small

and that the pressure would have to be too sharply localized, so that on the whole the theory that the finger pressure or forceps could produce it was unlikely. Pressure of finger he also rejects, for there was nothing for the finger to compress the plexus against. He comes finally to traction on the upper roots as the longest side of the triangle formed by the cords of the plexus, with lateral inclination of the head, as tending to increase the distance between the head and shoulder. He produced the paralysis in rabbits by pulling the head forcibly to one side. He showed that the amount of separation which occurred between the ends of the cut roots of the brachial plexus, when the shoulder was held down and the head carried to the opposite side with as much force as is used in ordinary labours, is as follows: The two upper cords, or fifth and sixth cervical, separated, from 26 to 28 mm., the third, or seventh cervical, only 12 mm., and the lower two, the eighth cervical and first dorsal only 8 mm. The point at which the rupture occurs is from a quarter to half an inch from the point of emergence from the spinal canal near the junction of the fifth and sixth cervical roots. Fibres of the suprascapular nerve always ruptured among the first.

Schoemaker also conducted experiments on cadavers with the plexus exposed, and could always tear the fifth and sixth cervical, but never the seventh and eighth. He also thought that the clavicle could cause pressure on the plexus by having it caught between the clavicle and first rib and spine. He was opposed to the theory that pressure from the fingers caused the injury. Kustner (second paper) and Landold also did experimental work and believed the injury due to traction. Stolper agreed in the main with Fieux and Schoemaker, but rejected the possibility of pressure on the plexus in breech cases, and believed that calvicular pressure might cause the paralysis. However, he believed that stretching was the main factor.

Other authors, such as Lovett[2], Carter[3], Walton[4], J. J. Thomas[5], Warrington and Jones[6], Stone[7], Bullard[8], Fairbanks[9], Taylor,[10] Osterhaus,[11] Frazier and Skillern[12], and Sharpe[13] and others, all believe in the theory of plexus injury due to traction, and support the known pathology as shown by operation and experiment.

Robinson[14] (1899) reports seventeen cases, in only one of which was the birth reported as normal. All the others had a definite history of the labour being tedious and difficult. In eleven the presentation was cranial; in three special mention was made of difficulty in delivering the arms; four others had forceps applied.

He states that J. E. Simpson has shown that the heads of boys are larger than the heads of girls, and therefore the heads of the latter would not dilate the way for the shoulders as well as the former. In his own series, thirteen babies out of seventeen were girls, which would bear out this theory that there was an insufficiently dilated canal for the shoulders and that they therefore caught, or were with difficulty delivered, and in so doing there was a strain put on the cords of the plexus.

T. T. Thomas[15] (1914), following Lange's theory, in an interesting theoretical discussion of the problem, based on a study of nine cases averaging 6·5 years, concludes that the paralysis is secondary to a primary traumatic dislocation of the shoulder occurring at birth, associated with a tear in the joint capsule, and a consequent involvement of the plexus in the exudate. He does not explain why the exudate always avoids the major portion of the plexus in the region of the shoulder joint, or why it practically always works its way at least two inches above the clavicle and picks out the junction of the fifth and sixth cervical nerves to produce the characteristic paralysis. This theory of his is not reasonable, nor is it based on clinical or pathologic evidence. Erb's point is small and it requires definite injury at this point to produce the characteristic paralysis, as well as injury above this point on the fifth cervical root to produce the paralysis of the supraspinatus and infraspinatus from trauma to the suprascapular nerve which comes off the fifth cervical just above or below Erb's point.

Ashhurst[16] in a recent paper defends the theory advanced by Lange and adopted by T. T. Thomas that the condition is not primarily due to injury to the brachial plexus, but is due to an unrecognized dislocation of the shoulder occurring at birth. I I might add that Thomas and Ashhurst so far are the only two individuals whose manual dexterity has been developed to such an extent that they can of all others determine this dislocation. Thomas, as you may remember, developed his original contribution as the result of the study of nine cases, seen late, that is, after several years, and Ashhurst reports about forty cases most of which were well by the early stages.

The statement is also made by Ashhurst that these cases all get well because no neurologist of his acquaintance has ever seen an adult with the condition. This is hardly a fact on which to base a scientific pathology. If a child is born with an obstetrical paralysis, the condition exists to a varying degree until death. Few cases every wholly recover and most carry always the well-known

mark of the so-called "policeman's tip" position through life, unless adequately treated for the deformity. Ashhurst considers the whole condition as to ætiology obscure, and cannot reconcile the resultant paralysis to the known distribution of the brachial plexus, overlooking the fact that many of the muscles have their supply from more than one spinal root. A further study and adequate observation on a sufficient number of cases with a willingness to accept proved pathology would at once clear the ground for him.

Pathology

There are generally two well-recognized types of paralysis seen. The more common one consists of a lesion which involves the fifth and sixth cervical roots and the suprascapular nerve and produces a paralysis of only the muscles of the upper arm, with the exception of the supinators. This type is known as the upper arm type as we have observed it in five hundred and thirteen cases. The less usual type, the so-called lower arm, or whole arm type, is the result of injury not only to the fifth and sixth cervical roots, but the seventh and eighth and possibly the first thoracic as well. Here the whole arm is flaccid; there is a wrist-drop and paralysis of the small muscles of the hand. There occasionally occurs the pure lower arm type of paralysis without any involvement of the upper cords of the plexus, the so-called Klumpke's paralysis, several cases having been reported by J. J. Thomas, Jolly, Guillemot, Seeligmuller, Thorburn, Raymond, Comby, and Danchez. These cases show a paralysis usually the result of stretching of the plexus from overextension of the arm in cases of face presentation, and due to injury to the lower cords of the plexus, namely, the seventh and eighth cervical and first dorsal roots. They may at times be bilateral. It is in this type that one often sees inequality of the pupils, owing to the fact that the sympathetic fibres from the deep cervical ganglionic plexus enter the spinal cord through the first dorsal and at times through the eighth cervical roots. Injury therefore to these roots leads to an unopposed action of the motor occuli nerve.

Pathologically, in the milder cases the stretching or tearing forces result in a greater or less degree of hæmorrhage or œdema into the nerve sheaths. In others there may be a rupture of the perineural sheath, accompanied by hæmorrhage into the substances of the nerve trunk, associated with the tearing apart or separation of the nerve fibres. This latter condition leads, of course, to per-

manently impaired function and the formation of scar tissue in the nerve track. In the more severe cases of the upper arm type there is a partial or complete division of the fifth and sixth cervical roots, which leads to a more permanent form of paralysis than usual, and the formation of a more extensive area of scar tissue.

The force producing these lesions is variable and so the lesions are variable. The nerve roots are often frayed out inside the sheath instead of being torn across evenly, and in this way the lesion may be incomplete at any given cross section of a nerve, but involves different fibres at different levels. This scar tissue contracts in time, and not only effectually prevents the regeneration of the nerves, but may by its contraction press on and destroy the few fibres which may have excaped the original injury.

The other type, known as the lower arm or whole arm type, is the result of either a lesion involving all the nerves of the plexus, or, in the distinctly lower arm type, in which the lower arm and hand are alone involved, the so-called Klumpke's paralysis, in which the lesion probably involves the eight cervical and first dorsal roots alone. This type generally results from traction applied in a breech case with the arm extended, or to traction in the axilla in a vertex presentation. It may be seen also in adults, when the first dorsal root is overstretched, as evidenced by some of the cases reported by T. T. Thomas. Pathologically, the conditions are similar to those seen in the other types, depending on the severity of the injury. No case in which operation has been performed has failed to show a definite pathologic lesion of the brachial plexus, definitely corresponding to the muscles involved.

Danyau (quoted by Stranksy[1]), in 1851, showed by necropsy that the nerves of the plexus had been torn and were surrounded and invaded by scar tissue. Practically all observers, especially those who have operated in these cases, have found definite changes in the plexus due to injury and scar tissue formation. Among these, for detailed study, may be mentioned Boyer[17], Fairbank, Warrington and Jones, Osterhaus, J. J. Thomas, Stone, Taylor, and Prout[18]. Prout's description of the pathology is classic and will be quoted freely as follows:

Prout states that the nerve sheath in any overstretching process must give way before the nerve itself as it supports the nerve. When the sheath is torn, as it always is in cases of birth palsy, the arterioles belonging to it and supported by it are ruptured, and a hæmorrhage into the substance of the nerve and its sheath results. These facts are of the greatest importance, since they determine

the ultimate extent and final character of the lesion. Were it not for the obstructive features of the repair process in the nerve sheath, we might expect a more or less complete recovery in the vast majority of cases.

Four pathologic specimens showed on study the following conditions: The usual seat of the lesion was at the junction of the fifth and sixth cervical nerves. The perineural sheath presented many old dense pigment deposits, the site of old hæmorrhages. In some portions the perineural sheath was buckled inward on the nerve fibres, strangulating them and preventing their regeneration. Evidences of strangulation were present not only at these points, but also in the nerve fibres underlying these pigment deposits. There was an obliteration of the myelin sheath above and below. In the more severe cases the strands of the plexus involved came to an abrupt termination in a mass representing an old organized hæmorrhage. In these cases there was a severing of the nerve fibres, which were often thrown into folds for some distance from the primary lesion. Repair of the nerve sheath takes place before the regeneration of the nerve fibres, and if this has buckled inward on the nerve bundles following relief of tension, the nerve fibres are inevitably going to be strangulated and their regeneration prevented.

Writer's Experiments

The writer, by numerous dissections on infantile cadavers, has shown that traction and forcible separation of the head and shoulder puts the upper cords, the fifth and sixth cervical roots of the brachial plexus, under dangerous tension. This tension is so great that the two upper cords stand out like violin strings. Any sudden force applied with the head bent to the side and the shoulder held would without question injure these cords. Further observation shows that forcible abduction and elevation of the arm and shoulder put the lower cords of the plexus, the eighth cervical and first thoracic on a stretch, and when much force is applied it may well lead to a tear, rupture, or other injury to these segments. This condition is seen in breech cases, with arms extended. It may also follow sudden strain when the arm is elevated, such as the so-called hostler's paralysis, caused by the sudden elevation and strain of the arm which occurs when a hostler holds a rearing horse. With the shoulder held and the head carried to one side, with the clavicle intact, considerable force was necessary to injure the plexus. The suprascapular nerve always snapped first, apparently for the

reason that it had not so much freedom of play as the others. Even with considerable force the fifth and sixth cervical nerves could not be completely torn across at Erb's point, but frayed out inside the sheath, following a partial tearing or rupture of the sheath, which always gave way first. In some cases there could be produced an evulsion from the spinal cord of the fifth and sixth cervical roots.

With the clavicle removed, the whole weight of the shoulder came practically directly on the plexus, and less force had to be exerted to cause an injury, which under these conditions was generally greater in extent, but presented the same general characteristics. It was most difficult to put the eight cervical and first thoracic roots on a stretch unless the arm was abducted or hyperextended with great force.

With the clavicle intact there was apparently always enough room, even with the arm elevated and hyperextended forcibly between the clavicle and plexus so that direct pressure from the intact clavicle on the plexus did not seem a possible cause of the paralytic condition. A fractured clavicle of course allows the weight of the shoulder to drag on the plexus, and so predisposes to greater injury from traction. Rotation of the head combined with forcible abduction apparently does not increase the degree of tension greatly, certainly not enough to cause additional damage. In no case, even with all the force I could apply with my hands, could I rupture the joint capsule, or even separate the humeral epiphysis. Neither could I dislocate the head of the humerus. The clavicle can be broken without great force, but fracture of the other bones which go to make up the shoulder joint is practically impossible. Most birth fractures occur in the clavicle, or in the humerus, at about the junction of its upper and middle third. Stone states in the experimental work which he did that the humeral epiphysis could be easily separated, but I failed to confirm this.

At birth the shaft of the humerus is nearly wholly ossified, but the two extremities are cartilaginous. The scapula at birth is largely osseous, with the exception of the glenoid fossa, the coracoid and acromial processes, and the posterior border and inferior angle, which are still cartilaginous. It is on account of these conditions that fractures in these regions at birth are practically non-existent. It is not possible to produce a paralysis of the Erb type by the fracture of any bone but the clavicle.

In order to get a clear idea as to what happened to an exudate

from a ruptured capsular ligament of the shoulder, in studying Lange's theory, I injected the shoulder joints of several infants with methylene blue, and then caused a rupture of the anterior portions of the joint capsule. The infants were then allowed to lie in a preserving solution on their backs for several weeks, following which time a dissection was made. In no case did the methylene blue go above the clavicle, but completely surrounded and invaded the plexus in the axilla. This would in life lead to a paralysis of the whole arm below the joint, but would in no way affect the nerves above the clavicle, and in no case would there be the typical picture of obstetric paralysis, that is, paralysis of the fifth and sixth cervical nerves. As I have stated before, why the exudate should leave the nerves alone in immediate proximity of the shoulder joint and seek out Erb's point, the junction of the fifth and sixth cervical segments, at least two or three inches above the clavicle, Lange, Thomas, and others have not made quite clear. It evidently does not happen. Why also should the suprascapular nerve always be involved, which generally arises from the fifth cervical at about Erb's point? One thing impressed me, and that was the evident vulnerability of the upper cords of the plexus under any degree of traction and I was surprised that the paralysis was not of much more frequent occurrence.

Roentgen-Ray findings. About two hundred cases have been *x*-rayed. These patients varied in age from two days to eighteen years. In only two cases had there been fracture, one of the clavicle and one of the upper third of the humerus. Both fractures had healed without incident.

A study of the roentgenograms taken in these cases shows the following conditions:

In the first year there is usually nothing seen of bony deformity. There may be a slight posterior sublaxation of the shoulder joint, but there is never any acromial deformity evident by roentgenogram or clinically. No case has been observed in which the epiphysis has been displaced primarily so far as could be seen by comparison with the normal shoulder. The epiphysis, as well as the shaft of the humerus, is always smaller than the unaffected side, which condition is undoubtedly due to atrophy from disuse. The scapula is practically always elevated and outwardly rotated, due apparently to the pull of the intact inward rotators and the levator anguli scapulæ.

As time goes on and the child gets older, one begins to see increasing evidences of bony deformity, occasionally more joint

subluxation than at first, increasing outward displacement and elevation of the scapula, and acromial deformity. The deformity of the acromion consists of a bending downward and forward or a hooking of its outer end, which, apparently, having no bony resistance to meet as normally in the head of the humerus, projects downward in front of the subluxated and inwardly rotated head. This hooking seems to vary directly with the degree of posterior subluxation and inward rotation of the humerus and tends to increase as the child gets older, provided subluxation is present. No case has been observed in which there has been a total subluxation or dislocation of the shoulder joint backward. The clavicle usually is shorter and its curves are more acute than its normal fellow.

Clinical Findings. When the child is first seen, if within a few days or weeks after birth, the following picture is classic. The arm lies limp at the side, extended and inwardly rotated, with complete inability to abduct, elevate, outwardly rotate or supinate. The muscles paralyzed in the typical upper arm type are as follows: Deltoid, supraspinatus, infraspinatus, teres minor, biceps, triceps, supinator longus, and occasionally the subscapula, the serratus magnus, coracobrachialis and supinator brevis. The arm cannot be actively flexed at the elbow, but as a rule the lower arm is not affected so far as flexion and extension of the wrist and flexion and extension of the fingers go.

The greater part of the motor nerve supply to these paralyzed muscles depends on one root alone, although fibres from more than one root, especially the sixth cervical, can be traced to individual muscles of the arm.

It should be noted that a number of these muscles have more than one source of supply. Expressed in terms of motion the condition is as follows:

Flexion of the elbow is carried out by the fifth cervical; extension of the elbow by the seventh cervical; pronation of the hand by the sixth cervical; supination of the hand by the fifth cervical; flexion of the wrist by the eighth cervical, and extension of the wrist by the seventh cervical.

In the upper arm type then, the nerves involved are the suprascapula, from the fifth cervical root and outer cord of plexus, going to the supraspinatus and infraspinatus muscles. The musculocutaneous from the fifth and sixth cervical roots and outer cord of the plexus, going to the coracobrachialis, biceps and brachialis anticus. The circumflex from the fifth and sixth, and possible the seventh and eighth and posterior cord of the plexus, going to the

deltoideus and teres minor. The musculospiralis from the fifth, sixth, and seventh, and also possibly some fibres from the eighth cervical and posterior cord of the plexus, going to the supinator longus and brevis, brachialis anticus, triceps, anceœus and extensors of hand.

The fact that in the upper arm type practically the only muscles supplied by the musculospiralis which are paralyzed below the elbow are the supinators goes to show that either the injury is not extensive or that the nerve root supply is well divided. No two diagrams of the brachial plexus among all that I studied were alike. The two best that I could find are, one from Cunningham's "Anatomy" and the other quite different is from Kocher.

In order to get this definite and constant paralytic muscle grouping, the injury would have to be located at about the junction of the fifth and sixth cervical nerve roots, just above the point of origin of the suprascapular nerve. This junction point is called Erb's point, from his classic description of the type of paralysis seen following injury at that point.

The inability to raise or abduct the arm at the shoulder is due to the paralysis of the deltoideus and supraspinatus. Outward rotation cannot be accomplished because of the paralysis of the infraspinatus and teres minor, and the arm cannot be internally rotated owing to the internal rotators, namely, the teres major, subscapularis and latissimus dorsi, being already fully contracted, due to lack of opposition.

The arm cannot be flexed at the elbow, owing to the paralysis or weakness of the biceps, brachialis anticus, coracobrachialis and supinator longus; and supination cannot be carried out owing partially to the inward rotation in which the arm is held and the weakness or paralysis of the biceps and supinator longus and brevis.

In regard to sensation, it may be stated that it has been impossible in the early cases to determine any changes from the normal, on account of the age of the patient.

During the first week, in the early cases, the child may cry if the arm is handled or moved, especially in abduction, but this soon disappears. In one or two cases there has been some swelling and tenderness noted by palpation over the plexus above the clavicle. This condition, however, apparently had no connection with the degree of paralysis present. The hand grasp is usually good and the child flexes and extends the wrist and fingers well. Occasionally there is a wrist drop present which in the upper arm cases was

only temporary. The later developments in the upper arm cases, as the child grows and gets older, with or without exercises and massage, are as follows: The persistence of the inward rotation and abduction deformity, the so-called policeman's tip position; the inability in most cases to fully or freely supinate; the inability to get the hand to the mouth without raising the elbow, due to inability outwardly to rotate; the inability to put the hand to the head or behind the back of the head.

In the lower arm type all these conditions hold besides the additional ones due to the paralytic conditions of the lower arm and hand, resulting generally in a useless dangle arm.

Atrophy of the muscles in these cases of obstetric paralysis is never very marked, except in some cases of the lower arm type. One practically never sees the extreme atrophy so noticeable in cases of infantile paralysis. This lack of marked atrophy is undoubtedly due to the fact that the nerve impulses are rarely fully blocked and that the muscles practically never, except in rare cases, wholly lose their entire enervation. Some normal nerve impulses pass through the scar tissue at the site of the lesions, owing to incomplete destruction or injury of the nerve, and so keep the muscle tone up to a certain point. There is always a definite shortening of the arm, however, in all cases, due probably as much to nerve injury as lack of use.

Subsequent Developments

Whole arm type, lower arm type. There were seen ninety-seven cases of this type in this present series. In this classification those cases which showed any nerve involvement beyond that usually shown by an injury of the fifth and sixth cervical roots were placed. These cases represented those injuries mainly to the whole of the plexus, or at least the seventh and eighth cervical and the first dorsal roots. Pupillary inequality and narrowing of the palpebral fissure were not unusual with this type. Occasional facial paralysis was present. Wrist-drop was the usual condition, associated with the usual inability to supinate and the additional inability to extend the lower arm. Paralysis of the flexors and extensors of the wrist and fingers were common, associated with paralysis and atrophy of the intrinsic muscles of the hand. Often the proximal phalanges are hyperextended, and the distal ones flexed, due to paralysis of the interossei or lumbricalis namus muscles. There is, of course, no power to grip and the fingers cannot be freely moved. There is

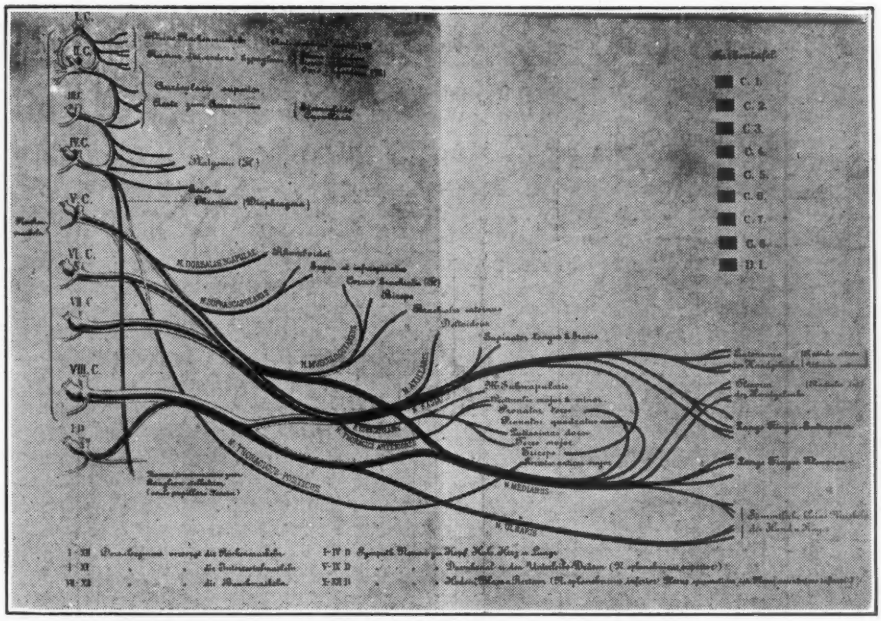
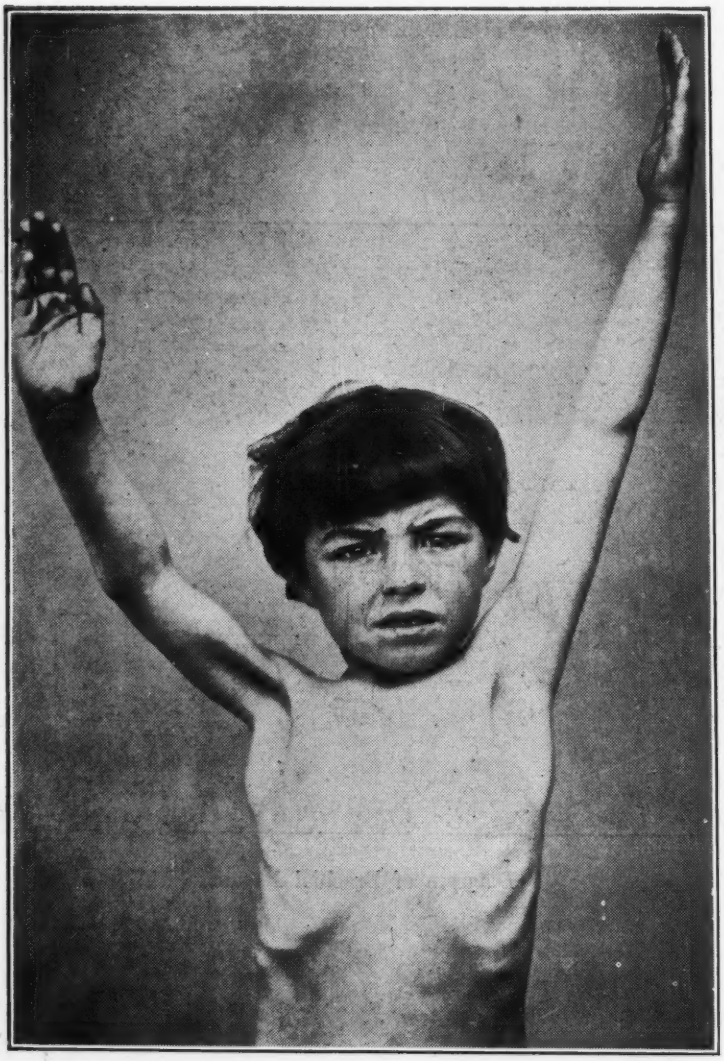
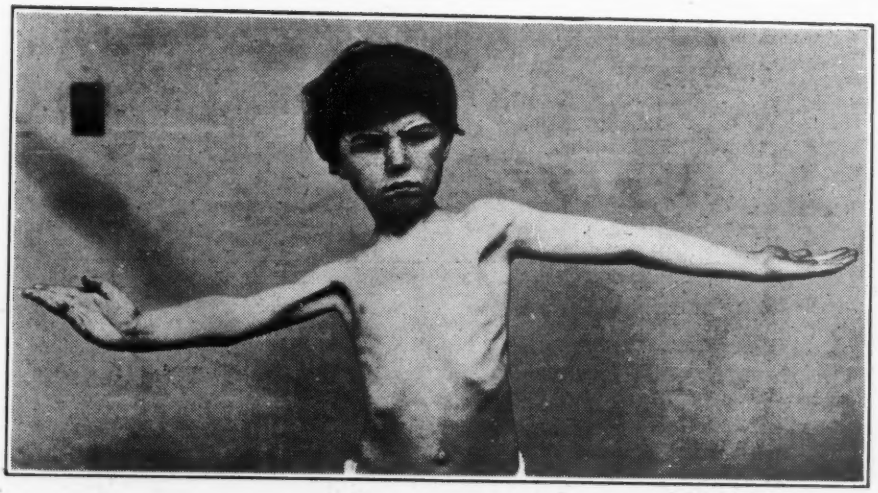


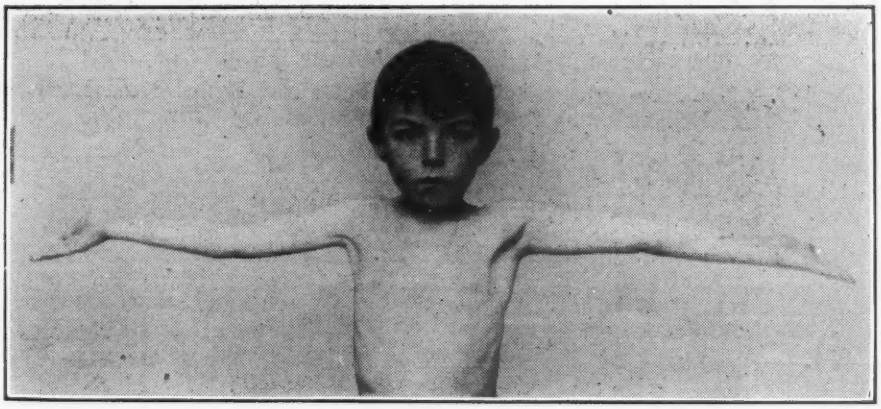
Diagram of Brachial Plexus



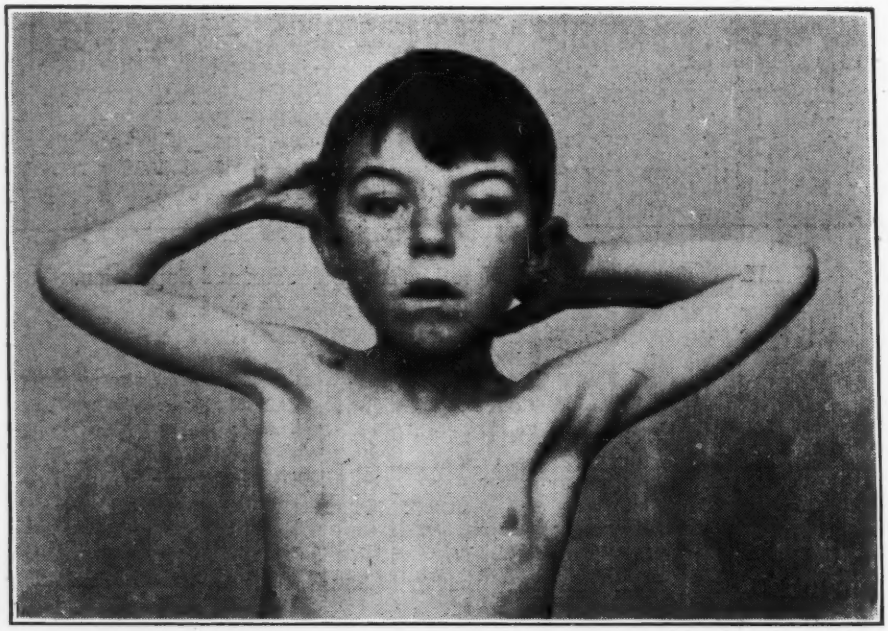
No. I.—Before Operation. Note Inability to Outwardly Rotate and Supinate Right Arm.



No. II.—Before Operation. Note Limited Abduction and Supination.



No. III.—After Operation. Note Free Abduction of Right Arm and Normal Supination.



No. IV.—After Operation. Note Ability to Put Hand Behind Head Easily, Demonstrating Free Outward Rotation—Right Arm.

usually ulnar displacement or abduction of the hand. These cases, almost without exception, represent severe tearing injuries to the roots of the plexus, and although some of the muscles may recover in part, particularly the upper arm and shoulder groups, the lower arm ones practically never recover from the point of view of function, even after attempted operative repair of the plexus. It is in these cases that sensation is more apt to be impaired than in the usual upper arm type. A not uncommon type seen is one showing simply a wrist-drop, associated with the usual picture of upper arm paralysis and evidence of injury to the fifth, sixth, and seventh cervical roots. These cases, as far as results go, should be classed with the simple upper arm type. Few cases have been recorded in which the two lower roots alone have been involved. These have been reported fully by J. J. Thomas.

The complications may be divided into two classes, early and late. The early complications are those accompanying the paralysis and present at birth. The following may be mentioned:

Facial paralysis is usually mild and on the same side as the paralyzed arm and is probably from forceps pressure on the facial nerve.

Fracture of clavicle is not rare.

Separation of epiphysis of the head of the humerus may occur, but no case is noted in this series; it might be grouped under the pseudoparalysis of D'Astros and Danchez.

Dislocation of the humerus sometimes is present, usually infraspinatus. This complication is not noted in this series, but is recorded by other observers.

Fracture of the upper third of the humerus may also occur.

As late complications the following may be mentioned:

Posterior subluxation of the humerus is common and due to contraction of unparalyzed pectoralis major, subscapularis and teres major.

Hooking of the acromion may occur, as had been already noted above.

Anterior subluxation of the humerus, due to the pull of the contracted pectoralis major and the stretching of the subscapularis, is not uncommon. Paralysis of the subscapula muscle is not rare.

Contraction of the biceps and the brachialis anticus, leading to some degree of permanent flexion deformity at the elbow and occasionally dislocation of the head of the radius, may occur, Persistence of marked loss of power in the triceps is common.

An analysis of the figures may be of interest. In the first

place, there is no reason to expect any difference in regard to the sex, unless one is ready to accept Simpson's theory that girls' heads, being smaller, and so not dilating the canal sufficiently, would subject them to a more difficult labor, and so to a greater percentage of occurrence of injury to the brachial plexus. These figures, representing by far the largest number of cases so far reported, and outnumbering all others reported by all observers, do not confirm his theory.

The right arm was affected three hundred and fifty-nine times and the left two hundred and forty, about 66 per cent. in favour of the right arm. This bears out Sharpe's figures in his series of fifty-six operative cases. Fourteen babies had both arms affected.

The types of paralysis differed, the most usual one being the so-called upper arm type, five hundred and thirteen being recorded, as against the so-called lower or whole arm type, in which, beside the fifth and sixth cervical cords being injured, the seventh and eighth cervical and first dorsal were injured. Of the latter type, ninety-seven cases were recorded. In fourteen cases with both arms affected, the lower or whole arm type of paralysis showed generally.

It has been conceded by practically all authors that a difficult labour was a predisposing factor in the causation of paralysis. In this series, five hundred and forty-one cases were definitely recorded as long, laborious and difficult; four hundred and sixty-two at least had ether and three hundred and sixty-one had forceps used; forty-four were apparently normal labours and two hundred and one were recorded in which the child was asphyxiated.

All the conditions noted above imply the application of force combined with greater muscular relaxation of the child, conditions peculiarly favourable for the production of such an injury. A moderately large number, it is recorded, had the head delivered naturally, but the shoulders stuck, and at that time force was applied.

In regard to the presentations, three hundred and twelve at least were vertex or face presentations and ninety-four were breech. The latter classification includes versions and footlings. In two hundred and eleven the position was not recorded, but a large majority of these were probably vertex. These figures do not bear out either Tubby or Sherren (quoted under Fairbank), who state that the paralysis occurs equally in head or breech presentations. Fairbank's own figures refute this also, for he reported in forty cases thirty-two vertex and seven breech. These figures cover

four hundred and six cases of the author's in which the presentation was definitely known.

The other conditions occurring at birth may be noted in the table and I want to add a word about only one of them, namely, that of unequal pupils. This condition is probably overlooked in some cases, and is a most important symptom, in that it means that through injury to the cervical sympathetic there may be definite injury to the plexus either of the lower cords, the eighth cervical or first dorsal, which have communicating bands with the cervical sympathetic, or injury in the spinal cord itself to the fibres of the sympathetic system. The prognosis in these cases is usually not so good as in those which do not show this sign.

Treatment

As to treatment, these cases at once resolve themselves into two divisions, namely, those to be treated with massage and exercises, principally those of the upper arm type; and those to be treated by operation on the plexus, usually those of the lower arm type. Unless the early treatment has been adequate, the upper arm type will also come to operation, not for plexus repair, but to correct contraction deformities. This operation, which I have devised, will be spoken of later.

At first, in order to prevent contraction of unparalyzed muscles, it seems best to put the arm at rest in such a position that the muscles cannot become contracted. This may be done by holding the arm in a plaster cast, or by the use of a light wire or aluminum splint, in an abducted, elevated and outwardly rotated position, with the hand supinated. This position can be maintained between massage and gymnastic treatments, and insures a better subsequent position of the arm. It also takes the drag off the paralyzed muscles, allowing them to regain their strength more quickly, and prevents subsequent shoulder joint deformity, such as subluxation and acromial hooking and overgrowth.

Massage and exercises are of the greatest importance and should be done daily if possible. It is most unwise to allow a child to become obsessed with the fact that it has an arm which cannot be used. Exercises which have been described in detail by J. J. Thomas are most satisfactory, and have been developed during the past twenty years in the neurologic department of the Children's Hospital. The treatment should be continued for several years at least, and if contractures develop in the subscapularis and

pectoralis major, they must be divided before any further range of action in the arm is to be hoped for.

In regard to the operation on the plexus in the usual upper arm type of case, it might be said that in the experience of this clinic it has not been found necessary. In the lower arm type of cases the situation is quite different, but it cannot be too strongly emphasized that no operation on the plexus will be of any great use in restoring functional activity to the arm, unless contracted and restricting muscles are divided, and careful after-treatment persisted in for a long period.

In regard to the operative treatment on the plexus in the lower arm type of case, it may be stated that it has been done a number of times without any benefit. The plexus in all cases was found to be so badly torn and so bound down and invaded by scar tissue that any kind of repair was impossible. In spite of the work done by A. S. Taylor, Stone, Fairbank, and others, there has been no case as yet which has shown an anatomic or physiologic cure, or even a marked improvement. This may be due to the fact that in in the first place the plexus was impossible to repair, and secondly, granted that the plexus repair was in part possible, the muscular contractions and joint deformities were not recognized and properly treated, without which the attempt to obtain plexus repair would be a waste of time and effort.

The following operation was devised, following suggestions made by Fairbank. It differs from Fairbank's operation principally in that the shoulder joint is not opened. Opening this joint leads to adhesions of the capsule, which are troublesome and fatal to the best functional results. In addition, I have found that complete division of the pectoralis major is always advisable, in that it is practically always tightly contracted, and so holds the arm adducted and prevents abduction and outward rotation. The subscapularis tendon can usually be easily found with the arm abducted and outwardly rotated after the division of the pectoralis major, and can be divided without opening the joint capsule.

Operation

An incision is made on the anterior aspect of the arm, beginning at the tip of the acromion and carried down to below the insertion of the pectoralis major. The cephalic vein is found generally in the outer edge of the wound and tied or drawn aside. The tendinous insertion of the pectoralis major is defined, raised on an

instrument, and divided all the way across near the bicipital groove. The pectoralis major muscle is then retracted inward out of the way, giving one a clear view of the axilla and shoulder joint. The arm should now be abducted fully and rotated outward as far as possible. Following the division of the pectoral, the range of motion in abduction will be found to be greatly increased. Outward rotation will, however, be somewhat limited. With the arm fully abducted and outwardly rotated, the insertion of the tendon of the subscapula is to be defined. This tendon is inserted on the lessor tuberosity of the humerus at its inner aspect, and its fibres run at right angles to those of the joint capsule, into which they merge. Just below the lower edge of the tendon may usually be found two or three small veins, running parallel to the lower edge of the tendon. The tendon of the coracobrachialis obscures the insertion of the subscapula tendon at times. It is then necessary to separate the origin of the coracobrachialis from the coracoid process by means of an osteotome, which gives one a much clearer field to see the insertion of the subscapula. The hole in which the surgeon is working is quite a deep one, and the tendon cannot be easily found unless the arm is in the position above described. The best way to divide the tendon is to pass under it some blunt instrument, and personally I have found a No. 18 French sound the most useful, not only because it is smooth and round and pointed, but also because its curve allows the point to present above the upper edge of the tendon and so defines it well. It is of the utmost importance that the shoulder joint should not be opened. The tendon of the subscapula should always be found, identified and lifted up before it is divided. Blind cuts along the capsule do more harm than good and should never be practised, even if following division of the capsule; the outward rotation is better. Eventually these capsular incisions lead to troublesome adhesions, and the results are never as good as when they are avoided. Following the division of the subscapula the outward rotation is perfectly free, as well as abduction. If at this stage there is still some subluxation of the head of the humerus which cannot be fully reduced, an osteotomy of the acromion should be done, and the loose distal piece either removed or tilted up so as to allow the head of the humerus to slip back into the glenoid. The wound is then closed with a few deep stitches and a continuous catgut stitch to the skin. No drainage is required. Very little bleeding usually takes place. The arm is then placed on a wire spint, which holds it elevated to or above the shoulder level, abducted and fully rotated outwardly

with the hand in full supination. At the end of ten days, massage, baking and exercises are begun, and continued daily, or at least four times a week. The splint should be worn night and day for at least three months, and daytime for at least three months longer.

The operation merely releases contractions, and gives the stretched and partly paralyzed muscles a chance to recover their tone and strength, and consequently the after-treatment is of the utmost importance. There have been thirty-two cases operated upon by this method.

The following questions have often been asked me in regard to this operation and while the number of cases operated upon is small, the results have been in the main so striking that I am going to try to answer them in detail.

1. What benefit, if any, has the operation caused?

In practically every case which has been operated on there has been free and full active outward rotation, as well as increased ability to elevate the arm at the shoulder, depending somewhat on the ability of the deltoid to regain its strength after long stretching and disuse, as well as more persistent residual paralysis in that muscle, which condition cannot be accurately determined beforehand, because of limitation of motion from contractures. Supination becomes either normal, or nearly so. The child can get the hand to the mouth easily, can put it on top of the head and behind the head, which in girls is all-important, so as to enable them to do their own hair. As a matter of fact, after following several hundred cases for several years, outward rotation and supination are never gained by the most persistent exercise treatment, even when stretching under ether is included.

2. What are the essentials for a successful operation?

A careful operation with free division of all contractures, and the utmost care in avoiding cutting the joint capsule. This cannot be too strongly insisted upon. Fixation in a splint and not plaster, which holds the arm elevated to above the shoulder level, abducted and outwardly rotated, with full supination. Fixation not continued for more than the time required to heal the wound, and then exercises, baking, and massage at least four times a week, wearing splint for at least six months.

3. On what cases should it be done?

The best results, as we see them, are on those cases who have had previous massage and muscle exercises, and who have some power in the deltoid and supraspinatus muscles. Treatment in all cases should be begun the first week of life, and the arm should be

put in the position of physiological rest—that is, abducted, elevated and outwardly rotated from the first, Cases should not have the arm tied to the side or across the chest, as so often is done at first, as this position encourages and develops contractures of the non-paralyzed muscles. Practically all cases, even those who have had no previous treatment, are distinctly improved, but the convalescence as far as active function goes is slower in those cases who have not had previous massage and exercise treatment. Any case which has contractures, even if only of the subscapula, is better if that contraction is divided. The operation does less harm than the contraction, and results in a more useful arm.

4. How long should the after-treatment be continued?

At least six months, wearing the splint night and day for at least three months and daytimes for three months more. Exercises, baking and massage at least four times a week.

5. What treatment before operation is necessary?

Every case should be given the benefit of the doubt, and should have a long course of at least a year of exercises and massage. In very young children it is better to wait until they are at least three years of age before operating. A splint should be worn during this period.

6. What can the child do after the operation that it could not do before?

The hand can be supinated, the arm can be outwardly rotated, and elevated to above the shoulder level, depending, as said above, on the strength in the deltoid. The hand can be put to the mouth naturally and on top and behind the head to do the hair. At first in some cases—that is, in the first six months or year—there is a persistent inability to adduct or inwardly rotate the arm. This clears up in time and unless the shoulder joint has been opened, is no cause of worry. Motion in the shoulder joint is always good in the end, unless the joint has been opened. A few cases where the joint has been opened have shown a persistent loss of motion in the shoulder and the arm has remained permanently abducted and outwardly rotated, with no motion in adduction. In these cases the free play of the scapula is of great benefit, and allows the use of the arm in a better position, at the expense of stretched rhomboids. The result, however, is not one to be desired, and can be avoided by leaving the capsule intact. Too long fixation following operation without exercises and massage will also lead to slow recovery of motion in rotation and adduction.

7. Is the gain permanent?

Yes. So far, at the end of about three years in some cases.

Prognosis

The prognosis in all upper arm type of cases is good, provided the case is watched from the start, and treatment property carried out. The patients are practically all able to raise the arm to the shoulder level and can use the hand and lower arm well, except for varying degrees of supination. Abduction and outward rotation are rarely regained without division of the contracted muscles, provided they have been allowed to contract.

In the lower arm type the outlook is not so good, although many of the patients regain use of the upper arm in spite of the persistent paralysis of the lower arm and hand. These cases should all be explored for repair of the plexus as far as possible, but even then very little hope can or should be held out to the parents. The general principles of treatment, however, should be carried out over a long period of time. Much can be done along orthopædic lines for these patients, and they should not be generally neglected as they have been in the past, with the statement that nothing can be done, or that they will get well of themselves.

Conclusion

Obstetric paralysis is due to stretching or tearing of the cervical roots of the plexus brachialis. It occurs in boys as frequently as in girls. It occurs more often on the right than on the left side.

The upper arm type is much more frequent than the lower arm type. It affects both arms very infrequently.

It is practically always associated with a difficult labour, in which ether and forceps have been used and force has been applied. Not uncommonly is the baby asphyxiated.

Head presentations show the larger percentage of occurrences of both types of cases.

It may rarely be associated with fracture of the clavicle but is not the result of a fractured humerus or a dislocated shoulder joint.

The prognosis for a useful arm is good in the upper arm type and bad in the lower arm type.

Table

Boys	298
Girls	319
	617

Right arm affected	359
Left arm affected	240
Both arms affected	14
Upper arm type	513
Lower or whole arm type	97
Labour difficult and long	541
Labor normal	44
Ether used	462
Forceps used	361
Asphyxiation of child	201
Presentation.	
Head (including face)	312
Breech (including foot and version)	94
Position not known	211
Severe operation	32

BIBLIOGRAPHY

1. STRANSKY.—*Centralbl. fuer die Grenzgeb. der Med. and Chir.*, 1902, with complete bibliography to date, 1902.

2. LOVETT.—"The Surgical Aspect of the Paralysis of New-Born Children." *Bost. Med. and Surg. Jour.*, July 7th, 1892.

3. CARTER.—"Obstetrical Paralysis, with Reference Especially to the Pathology and Etiology." *Bost. Med. and Surg. Jour.*, May 4th, 1893, cxxviii, No. 18.

4. WALTON.—"The Etiology of Obstetrical Paralysis." *Bost. Med. and Surg. Jour.*, December 24th, 1896, cxxvii.

5. THOMAS.—"Two Cases of Bilateral Birth Paralysis of the Lower Arm Type," with Bibliography. *Bost. Med. and Surg. Jour.*, October 19th, 1905, cliii, No. 16.

6. WARRINGTON and JONES.—"Paralysis of the Brachial Plexus." *Lancet*, December 15th, 1906.

7. STONE, J. S.—"Injuries about the Shoulder Joint at Birth." *Bost. Med. and Surg. Jour.*, March 8th, 1900, cxiii, No. 11.

8. BULLARD.—"Obstetric Paralysis." *Am. Jour. of Med. Sc.*, July, 1907.

9. FAIRBANK.—"Birth Palsy; Subluxation of the Shoulder-Joint in Infants and Young Children." *Lancet*, May 3rd, 1913.

10. TAYLOR.—"Results from the Surgical Treatment of Brachial Birth Palsy." *Jour. Amer. Med. Assoc.*, January 12th, 1907, xlviii, No. 2.

11. OSTERHAUS.—"Obstetrical Paralysis." *New York Med. Jour.*, November 7th, 1908.

12. FRAZIER and SKILLERN.—"Supraclavicular Subcutaneous Lesions of the Brachial Plexus not Associated with Skeletal Injuries." *Jour. Amer. Med. Assoc.*, December 16th, 1911, lvii, No. 25.

13. SHARPE.—"The Operative Treatment of Brachial Plexus Paralysis." *Jour. Amer. Med. Assoc.*, March 18th, 1916, lxvi, No. 12.

14. ROBINSON, H. B.—"Traumatic Birth Paralysis of the Upper Extremity." *St. Thomas Hospital Reports*, 1899, xxvi.

15. THOMAS.—"The Relation of Posterior Subluxations of the Shoulder Joint to Obstetrical Palsy of the Upper Extremity." *Ann. Surg.*, 1914.

16. ASHHURST, ASTLEY P. C.—*Annals of Surgery*, January, 1918, lxvii, page 25.

17. BOYER, G. F.—*Proc. Roy. Med. Soc.*, Neurol. Sect., 1913, page 31.

18. TAYLOR, A. S.—"Results from the Surgical Treatment of Brachial Birth Palsy." *Jour. Am. Med. Assoc.*, 1907, xlviii, 96.

THE TREATMENT OF EMPYEMA IN LOBAR PNEUMONIA BY EARLY ASPIRATION

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THE usual course of empyema in lobar pneumonia is something of which neither the physician nor surgeon has any great reason to be proud. The reference is to the ordinary form and not the special varieties which were seen so frequently in the army hospitals in the United States during the war. On the medical side we have been to blame because in many cases the diagnosis is made very late and after pus has been present not infrequently for weeks. On the surgical side, the usual management left much to be desired, and it is to be hoped that a better technique will be one result of the experience of the war.

Ask a number of medical men whether they regard empyema in lobar pneumonia as a complication or a sequel and the replies will probably be divided. In a few cases it is a sequel, but this is rare, and in the great majority it is a complication, that is, the empyema is present before the pneumonia has run its course. This means that we should endeavour to recognize the presence of empyema before the pneumonia is ended or at any rate within a few days after this. The usual average of time taken for the recognition of empyema is probably weeks instead of days.

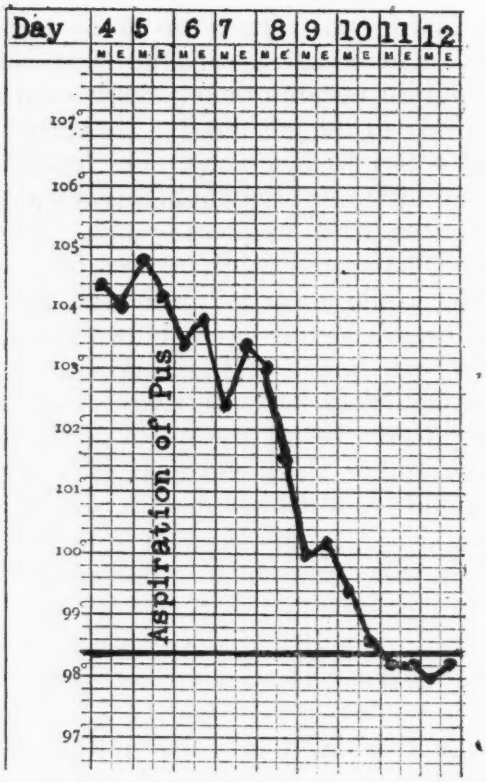
For some years I have been interested in the possibility of early recognition of empyema and its prevention, one might say, by immediate tapping. In place of the word prevention one might say better, cutting short any extension and preventing the need of operation for drainage. The following is an account of such a successful result.

The patient was a white male aged forty-six, admitted to my service in the Pennsylvania Hospital on August 30th, 1919, complaining of cough and severe pain in the chest. He was a Russian who spoke very little English and we had difficulty in getting any account of his past history.

His present illness began on August 27th with severe pain in

the chest, cough, and fever. There was no history of a chill or any note of exposure. The cough had come on in paroxysms and he states that the sputum became bloody very soon after the onset. The pain was entirely in the left chest. He states that he had felt feverish and had a severe headache. On admission the patient presented the usual picture of pneumonia and was very toxic. He had marked dyspnœa with frequent cough. The chest showed marked diminished expansion in the lower left side. There was involvement of the whole lower left lobe with marked dulness on percussion. The breath sounds were blowing and had a definite tubular character with fine crackling râles at the left base. In the upper part of the lower left lobe there were râles of a coarser character. Over an area below the angle of the scapula the percussion note seemed duller, there was more resistance on percussion and the breath sounds were rather distant and without any rales. The vocal fremitus over this area seemed to be decreased. On the day after admission, which was the fifth day of the disease, a needle was inserted at this point and purulent fluid was obtained. The fluid was not very thick and had somewhat of a greenish colour. Smears showed many pus cells, but no organisms could be found. Cultures were made but remained negative. The patient was very ill for the following two days, and the sputum was still bloody, but on the eighth day the temperature began to fall and reached normal on the tenth day of the disease. The leucocytes were 22,000 to 23,000. Towards the end of the febrile period the note over the lower left lobe became tympanitic, the tubular quality of the breath sounds lessened and a considerable number of râles, mainly of the bubbling quality, were heard. Over the area from which the fluid was obtained the clearing was rather slow. Vocal fremitus was perhaps slightly less marked, and on discharge there was some evidence of a slight amount of thickened pleura. The patient was discharged in good condition on the twentieth day after the onset, and has remained well since. The sputum was negative for tubercle bacilli and the Wassermann test was negative.

Remarks.—We have in this case the presence of pus in the pleural cavity on the fifth day of the attack of pneumonia, aspiration being done on the fifth day, and after this there was apparently complete absorption of any fluid remaining. It is evident that we cannot be certain that absorption might not have occurred without the aspiration. The patient might have been perfectly well able to take care of the infection himself and it is quite possible that this is a fairly common happening in lobar pneumonia. If so, it is



TEMPERATURE CHART

rarely recognized. My own feeling is, however, that it is more probable that the condition would have gone on with steady increase in the amount of fluid present. The fact that no organisms could be found in smears from the pus and that the cultures were negative may be of some significance. It may mean that the patient's resistance was so strong that any organisms in the pleural fluid were rapidly killed.

I have no intention of putting forward the view that in every case of empyema early tapping will result in a "cure", but there are a certain number of these cases in which it does, and it is important for us to be on the watch for them, recognize them early and treat them promptly. Every year I see two or three cases much like this one and with the same satisfactory result. This is not invariable and in a certain number of cases in which tapping is done early the process extends and subsequently the pus has to be drained. However, if one saves only an occasional patient from operation, it is certainly worth while.

This early recognition has to be made by careful study of the physical signs. The *x*-rays do not aid as the lung is consolidated and the collection of pus does not make any difference in the shadows. A careful study over small areas by light percussion and patient auscultation is the greatest help in the early recognition of these small collections of pus. The breath sounds over the area of fluid, which at this stage is small, are less clear and the voice sounds show alteration which is sharply limited. In all the cases in which pus has been obtained early, it has been thinner than is usually the case at a later stage.

The question comes up as to how long we should delay drainage and continue tapping if the presence of pus is discovered early. My answer to this is only a few days. In all the successful cases that I have seen the absorption was rapid and in the unsuccessful cases the increase was very evident. In this latter event the resort to surgery should not be delayed.

TWENTY-FIVE years ago comparatively little was known of tropical diseases; now schools of tropical medicine exist in many of the world's universities. Investigators have been sent to the tropics from every active nation. The discoveries have been valuable and have thrown light upon things unknown in diseases of temperate climates. The destruction of mosquitoes, and the consequent prevention of mosquito-borne diseases such as yellow fever and malaria; treatment by arseno-benzol which destroys the spirochætes of relapsing fever, syphilis, and other diseases of similar causation; the exhibition of ipecac in the alleviation of amœbic dysentery; these are all instances of efficient, direct and specific action that are not easily paralleled in the every day control of those diseases not usually known as tropical. Most of the known causes of disease in temperate climates are bacteria. Some of the diseases of tropical countries are also caused by bacteria; but the best known are due to protozoan parasites. Some of them are associated with organisms of uncertain position, and of a few the cause is still unknown. The protozoan parasites are much larger than bacteria, and are often actively motile. Their visibility is doubtless one of the reasons which has caused so much work to be done in tropical diseases, and which has contributed to the success of that work; it was one reason which led Ehrlich to use trypanosomes for the researches which gave "606".

BLOOD TRANSFUSION

By Charles K. P. Henry, F.A.C.S.

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SINCE Richard Lower of England in 1665 experimentally in animals successfully practised blood transfusion, and Jean Denys of France two years later successfully transfused sheep's blood into man, the procedure has been a source of constant experiment and progress. Like other great steps in therapeutic procedure it has had its rise and fall, and to-day it has reached the highest point of efficiency and use, largely because of its great purpose, carried to successful fulfilment, of saving thousands of lives in the Great War.

A few days since a surgeon from a Canadian casualty clearing station stated that had the methods and knowledge necessary for blood transfusion been as universally available in the first two years of the war as in the two later ones, "thousands of lives more would have been saved".

The literature of the last few years is replete with papers on blood transfusion, and an effort will be made in this paper to show the progress and the status of blood transfusion in present day therapy.

Indications for Blood Transfusion

Leisrink in 1876 said, "Transfusion is indicated in all those pathological conditions when the blood in quantity and quality is so altered that it is unfit to fulfil its physiological duties."[1] Such deficiences may be summarized as:

(*a*) Deficiency in quantity:
For example: Post-hæmorrhagic anæmias and secondary anæmias from chronic sepsis.

(*b*) Deficiency in quality:
For example: Pernicious anæmia; hæmolytic jaundice.

(*c*) Deficiency in function of clotting, from deficient amount of proper thrombo-plastic substances.
For example: Hæmophilia.

Correspondingly blood transfusion is indicated:

(*a*) To replace loss of blood—due to injury or disease.
(*b*) To stimulate the hæmatopoietic organs.
(*c*) To add a thrombo-plastic substance in those cases of prolonged coagulation.

The first indication will necessarily be the most common, and in civil as in military surgery, its greatest successful field is that of post-traumatic anæmia, either with or without surgical shock. Blood transfusion will, as does no other procedure, quickly restore the balance necessary to the circulation, for fluid bulk is a first essential to a proper circulation. If patients are to recover following hæmorrhage there must be 65 per cent. of their normal blood volume available and at least 25 per cent. of their total hæmoglobin.[2] For an adult male of average weight it is estimated that 5,000 to 6,000 c.c. represents the total blood bulk. Following hæmorrhage the hæmoglobin is estimated and it may be, for an example, 80 per cent. Then a 500 c.c. infusion of saline is given and the hæmoglobin estimated, when it will be 70 per cent., a loss of 10 per cent. on account of dilution. Therefore it may be reasoned that 10 is to 80 as 500 is to x, or the total volume of blood. In the example given, x equals 4,000 c.c. or 3,500 c.c. of blood prior to the saline infusion. If it was estimated that the patient had an average total blood content of 6,000 c.c., he had, after hæmorrhage only 35/60 of normal capacity or 57 per cent., and needed a blood transfusion. His actual total hæmoglobin would be three-fourths of the relative hæmoglobin which was 70 per cent. for 4,000 c.c. of blood, *i.e.*, 52½ per cent. As early as 1869 Hicks said "the want of success in transfusion lies in the postponement of the operation until too late a period". DePage found that with wounded soldiers, when the red blood cells fell below 4,500,000 in three hours; 4,000,000 in eight hours, or 3,500,000 in the first twelve hours, the patient would probably die unless transfused. It has also been shown that permanent degenerative changes occur in the organism when the exsanguinated condition persists for more than a few hours. Surgical shock without hæmorrhage can often be diagnosed by such estimation and it is generally conceded that blood transfusion, in cases of shock unaccompanied by hæmorrhage, is not beneficial.'[4]

The infusion of saline and gum arabic solutions are substitutes for blood transfusion, and fail of its full benefit because they are, especially the former, rapidly lost from the blood vascular channels, they fail to increase the oxygen carrying function of the blood, and they do not increase the hæmostatic or hæmatopoietic functions

of the blood. Crabtree[3] states that an hour's heat, with morphia when required, and a 700 c.c. blood transfusion, make hopeless looking cases safe operative risks, and second transfusion after operation, of 500 c.c. restores the patient to his pre-operative condition. Blood transfusion was used if the patient failed to rally in half an hour under rest, heat, fixation of fracture, and morphia. The use of gum solutions was of secondary value, many failed to improve till followed by blood transfusion. Soda bicarbonate solutions 2 per cent. strength in 6 per cent. glucose was found of great value in gas gangrene cases associated with severe vomiting and was often used in connection with the infusion of blood. It is to be noted that prolonged boiling of the soda bicarbonate solution may convert it into the carbonate and subcutaneous injection will lead to considerable necrosis of tissue.

Of the commoner conditions met with where blood transfusion is used for actual hæmorrhage: traumatism, gastric and duodenal ulcer, postpartum hæmorrhage, ruptured ectopic pregnancy, typhoid hæmorrhage and bleeding hæmorrhoids are the most commonly met with. In our own series of fifty-four cases the conditions calling for blood transfusion in this group were: intestinal and gastric hæmorrhages, fifteen cases; postpartum and post-abortion, eight cases; secondary anæmias, six; and of these twenty-nine cases, five were transfused preparatory to operation. Many dangerous operative procedures may be safely conducted following a transfusion, and post-operative convalescence shortened by transfusion before or after or both. Post-operative hæmorrhage in cases of jaundice may be forestalled, lessened, and recurrence prevented by blood transfusion. Post-operative shock, anæmia and prostration is lessened or prevented by the same procedure. Our series gives cases where this procedure was shown to be helpful. In acute sepsis, or septicæmia, blood transfusion seems to be of no value.[3] In chronic sepsis, and secondary anæmia due to chronic sepsis it is invariably beneficial. Hooker[24] and others have shown the value of infusions of blood from donors previously treated to increase their immunity. In the treatment of pneumonia and epidemic influenza blood transfusion from donors who had recovered from influenza and pneumonia was not productive of appreciable results. I used two such donors in one case of influenza and pneumonia where temporary benefit, if any, alone resulted.

In chronic infections accompanied by anæmia the benefit of transfusion would appear to be partly due to the increase of resistance to infection, and the presence of antitoxic and bactericidal

properties in the infused blood from the healthy donor.[2] In one case of postpartum septicæmia, No. 10 in our series, the temperature fell to normal in thirteen days, and the pulse dropped ten to twenty beats on each of three successive days after transfusion of 750 c.c. of blood. In a case of prolonged suppuration due to psoas abscess and staphylococcus arthritis of the elbow, a marked and continued improvement followed transfusion in case No. 9. Other indications for blood transfusion are many of the poisonings and intoxications as benzol[21], illuminating gas, etc.[6]

Limitations of Blood Transfusion

The therapy is limited chiefly by the necessity of making proper blood tests of recipient and donor and by the technical skill and apparatus needed to carry out the operation. Of the former, little need be said. Since Jansky in 1907 discovered that all individuals early in the first few weeks of life fall into one of four blood groups, of which each has its iso-agglutinins as shown in 1900–1901 by Landsteiner and Shattuck, it was only necessary for Moss to add that hæmolysis could not occur without prior agglutination of the red blood cells to enable us to formulate proper tests for safe blood transfusion. Any laboratory, of course, can test recipient and donor, and as long as a donor from the recipient's group, or from the universal donor group, No. 4, is used, no agglutination or hæmolysis may be expected. Lee[7] has shown how simple the test may be made, using a glass slide, a drop of serum separated from one or two c.c. of blood of the recipient mixed with a drop or two of a cell suspension obtained from letting fall three drops of blood from the donor's ear into 1 c.c. of 2 per cent. soda citrate solution. If no agglutination occurs, the operation may be safely carried out. For town or country practice, test papers may readily be obtained from a laboratory, made by saturating heavy white paper with No. 2 and No. 3 sera on which a drop of the cell suspension of the blood to be tested is dropped.[8] These papers can be kept in oiled or waterproof paper and retain their activity for a long time. It is to be expected that the various leading pharmaceutical firms now dealing in vaccines and sera will have these available as the demand grows for a quick, simple and safe test.

In a few cases where repeated transfusions have been done, using different donors, it has been found that late in the series one may encounter an agglutination reaction, and the test for the

prospective donor should be repeated each time to avoid a dangerous hæmolytic reaction.[9]

The storing of blood in which clotting has been prevented by the citrate method enables blood transfusion to be carried out at a distance from the donor, and such stored blood may be used up to four weeks after withdrawal.[10] In fact, the utility of such stored blood is limited only by the life of the red blood cell. This has been found to be thirty days or more[11], and the determination of the length of life of the transfused red cell has been accomplished by the agglutination reaction of red blood cells withdrawn from the recipient from time to time in this period with known sera of the various four blood groups. The initial improvement following transfusion varies in our series for the first twenty-four hours from 300,000 to 2,000,000 red blood cells and 10 per cent. to 20 per cent. hæmoglobin. Between four and five days after transfusion a secondary rise in hæmoglobin and red blood cells occurs from stimulation of the hæmatopoietic apparatus. Consequently repeated transfusions of moderate amounts, 500 to 750 c.c. at intervals of five to seven days, give the best results in chronic anæmias, like pernicious anæmias and chronic sepsis like osteo-myelitis.

Methods of Blood Transfusion

The direct method of transfusion has passed. In 1918-1919 we can say that a vast majority of all transfusions have been done by the citrate method. Between this and the period of direct transfusion, the syringe method, perfected by Lindeman, the paraffin tube method of Kimpton, Brown, Vincent and others, and other indirect methods had their warm advocates. Up till December, 1917, the Kimpton, Brown and Vincent tubes were used by me with satisfaction. Since then the citrate method has been entirely used with two exceptions in January, 1918. Of the largest number of transfusions reported by any one author, 1,036, we find 1,001 done by this method, all done since December, 1915, at the Mayo Clinic[1]. On November 14th, 1914, Professor L. Agote, of Buenos Aires, is said to have done the first citrate transfusion in a case of placenta prævia, abstracted in *Surgery, Gynæcology and Obstetrics*, February, 1919, page 153. The method was suggested in 1914 by Richard Lewisohn of New York, and he reported twenty-two cases in 1915[12].

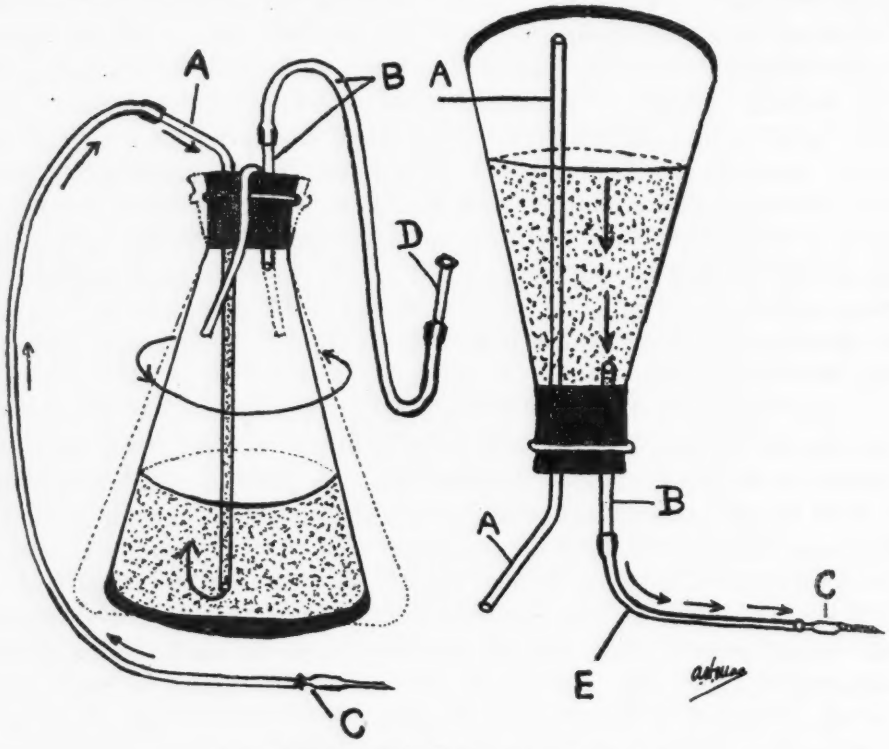
It is the simplest available method to-day, is the most adaptable to war conditions, and alone permits of the obtaining blood,

transportation of it and storage until the need for it occurs. It answers all requirements as to facility and simplicity of technique and apparatus, rapidity, efficacy and security. There are numerous methods and apparatus used to prevent clotting of the blood by the citrate method, but the simplest and handiest is in my opinion that shown in *Surgery, Gynæcology and Obstetrics*, March, 1919, page 265, by Pemberton[1]. This method has been used in the Montreal General Hospital since December, 1917, and answers well. The glassware necessary is cheap and easily obtained, and the Kaliski (gauge 11) needle is large and easily introduced, save into veins of exsanguinated patients when dissection of a vein is at times necessary. The outfit is sterilized immediately after use and the autoclaved saline and distilled water is always ready. Prior to transfusion the distilled water has added to it the necessary quantity of citrate (36 grains to 120 c.c. of water), and is boiled for three minutes. The toxic dose of citrate is 0·3 grams per kilo, and in the above 120 c.c. there is not more than 2·34 grams, or about half the toxic dose.

For each 220 c.c. of blood required there is needed 30 c.c. of the above solution. This must be freshly prepared each time as citrate in solution rapidly deteriorates. A piece of rubber tubing is held in place about the arm, not tight enough to impede arterial flow and the vein is fixed by passing a fine cambric needle through the skin and transfixing it close to the skin by Watson's method[13]. A Kaliski's needle is then entered a quarter of an inch proximal to the transfixion needle and enters the vein beneath the needle, pointing towards the hand. The Kaliski needle has attached to it an 8-inch length of firm rubber tubing, about three sixteenths of an inch calibre inside. As soon as it enters the vein the blood flows and is conducted into the receiving beaker where it is mixed by a glass rod with the citrate solution. To prevent the tendency to clot in the needle or tubing I have recently filled the needle and tubing with citrate solution and clamped the end of the tube. After entering the skin, the clamp is removed and the needle pushed into the vein when the blood forces out the citrate, and is not exposed to air till it falls into the beaker. It is thus easy with one needle and tube to secure 440 to 660 c.c. or more of blood with a uniform steady flow from a single puncture. The mixed blood and citrate is then run into the patient's vein with any ordinary salvarsan set. A simple graduated cylinder with tubing, glass tell-tale and needle is quite satisfactory. The blood may be carried to the ward or given elsewhere than where obtained. If to be kept

in storage it is preferable to use a flask with a cork and two glass tubes, one being used for suction either by mouth or by pump or bulb. The flask will contain the necessary citrate in solution and can then readily be used for the recipient by simply pumping air into the flask and forcing the blood out into the recipient's vein.

Farr shows a litre Erlemeyer flask adapted for this purpose.[14]



C—Adapted to fit Luer or record needle.
D—Mouthpiece for suction.

The Robertson pressure bottle was used in the Canadian army, and though not as simple as the gravity method of collecting blood, has the advantage of being used for administration as well. By syphon action, using two bottles, a very simple and easily regulated apparatus can be made.[15]

It would be useless to mention all the methods of blood citration and the technique, or to recite the various authors who advocate it as preferable in military surgery.

Contrary perhaps to expectation, the addition of citrated blood does not lower the time of clotting for the recipient. The reverse is true. In no case has it been found that the time has

been lengthened, and the addition of the blood with its additional thrombo-plastic substances has decreased the time of coagulation.[15] In bleeders this is most marked and a case is reported when the coagulation time was reduced from two and one-half hours to fifteen minutes by a citrated blood transfusion.[12]

The Results of Blood Transfusion

No uncertainty is evidenced by any operators who have had experience with the infusion of blood. It is the quickest and best method of resuscitation following hæmorrhage. With the aid of heat, rest, quiet, relief from pain by morphia, and fluids by mouth or rectum, or transfusion of blood, cases that appeared hopeless on admission to casualty clearing stations rapidly improved, recovered from shock, underwent major operations, and were evacuated to the base in good condition. Equally striking results in civil surgery have been obtained in ordinary industrial and city traumatic cases of hæmorrhage. So too with anæmia due to disease. case No. 2 in our series was comatose, showed œdema of the extremities, was lemon yellow in colour, blood count was: red blood cells, 1,500,000; white blood cells, 53,600; hæmoglobin, 26 per cent. when transfused February 22nd, 1916. He was transfused again on March 2nd, operated on for bleeding hæmorrhoids March 4th, and discharged walking from the hospital a week later. His blood count was then nearly 70 per cent. of normal.

In pernicious anæmia the improvement is usually prompt, and in most cases, thirteen out of fifteen in our series, an improvement in general well-being as well as in the blood count was noted. Even after the first week most of the cases gave evidence of having received a stimulus to their blood forming apparatus, as evidenced by an increase in the red blood cell count. Pernicious anæmia cases require from three to five transfusions at weekly intervals and then about once a month. Even with a prolonged series of transfusions as carried out by McClure, the fatal outcome is at best but postponed.[23]

Chronic sepsis offers a favorable field for transfusion, and the improvement in the blood picture is always accompanied by an improvement in the local septic or inflammatory condition. Chronic bone disease, psoas abscess, tuberculosis of bone and lungs, offer cases of anæmia that have been found to be benefitted by blood transfusion. The range of temperature is lessened and the body metabolism is slowed as evidenced by a fall in pulse rate, respira-

tory activity and a lessening of the elimination of pigment through the urine.[17]

Reactions Following Blood Transfusion

These are often disagreeable and annoying to the patient, but are temporary, not dangerous, and have no deleterious effect. They range from a slight chilliness and nausea to a rise of 2½ degrees or more with a rigour and severe vomiting. Headache, urticaria, and at times slight œdema of the eyelids, lips, tongue, and even glottis occur. The percentage in all transfusions varies somewhat with the method. Lindeman reports two hundred and fourteen consecutive transfusions without a chill by the syringe method.[18] On a large series of citrate transfusions the frequency of reactions is between 16 per cent. and 20 per cent.; nearer the latter. In our last twenty-seven cases, done by the citrate method, we have had six marked reactions, chill and temperature elevation of 2½ degrees or more, sometimes with vomiting; in three cases a slight reaction evidenced by chilliness, headache or nausea, but without any rise of temperature; or about 33⅓ per cent. of reactions. By the paraffin tube methods we had four reactions with twenty-three cases, and three cases of agglutination from use of improper donors. The Mayo Clinic[1] reports 21 per cent. of reactions, and V. C. Hunt 18·7 per cent.[19] The above reactions must not be confused with the agglutination or hæmolytic reactions due to improper blood mixing. In one of our cases a very marked hæmolytic reaction occurred which, in a seriously ill patient, very materially hastened his end. In one other, where a donor of the same group could not be obtained, we used a donor whose red blood cells were not agglutinated by the recipient's serum and yet we got evidence of hæmolysis, hæmaglobinuria, etc. This patient had pernicious anæmia and had been transfused before. In our third case, No. 44, the laboratory made an error as we got evidence of agglutination as soon as 125 c.c. of blood had entered her veins, when the patient became faint, complained of ringing in her ears, showed marked cyanosis, had severe headache, some clonic spasms and rapid pulse. The urine afterwards showed blood. A second test showed the patient was in group 4, not in group 2, as previously reported, and as was her donor.

Frequently repeated transfusions will show increasing difficulty in matching bloods, and reactions of hæmolysis may occur from the formation of iso-hæmolysins.[9] It has been worked out

that the cause of the reactions we meet with are not due to the citrate used nor to the method of transfusion *per se*. Reactions are less frequent with increased dexterity, and in those citrate transfusions which went smoothest and most rapidly we had no reactions. I believe that the less traumatism the blood receives in the mixing the fewer are the reactions.

Of the blood itself it is proven that the plasma has no toxic effect, as when the cells are washed and the plasma removed, reactions still occur. The toxicity resides in the cellular elements, one element certainly in the blood platelets. The washed whole cell content is toxic even for the person from whom the blood was withdrawn. The re-introduction of blood may cause a marked reaction in the individual from whom it was withdrawn.[9]

The average frequency for reactions to occur may be put at a minimum of 20 per cent. and a maximum of 40 per cent. It does not appear at present possible to state whether patients who have no reaction do better or derive more benefit than those who have a typical reaction. The time of the appearance of the symptoms that indicate agglutination or hæmolysis is quite apart from the time of the appearance of the symptoms of an ordinary post-transfusion reaction.

In the former these are quick in their onset, occurring within the first few minutes of transfusion, usually beginning as soon as 50 or 75 c.c. of blood have been introduced into the recipient, and rapidly increasing in intensity if the transfusion is carried on. Hypodermics of adrenaline and of atropine help to check the ill results. In the latter they rarely begin till fifteen minutes after the completion of the transfusion, and usually only some time after the patient returns to the ward, or within the first hour.

The transfusion of blood can be carried out by the citrate method in twenty to thirty minutes and 660 c.c. of blood given readily in this time.

In infants the transfusion is often carried out through the anterior fontanelle into the superior longitudinal sinus. Personally I have not transfused an infant this way, but have administered glucose saline by this route in a marasmic infant. A 5, 10 or 15 c.c. Luer syringe with a 21 or 23 gauge needle is sufficient, or the safer, if somewhat more cumbersome needle of A. Goldbloom may be used. This is fitted with a set screw to enable the operator to gauge the depth of the needle.[16]

In most cases of hæmorrhage of the new-born it is sufficient to inject 10 c.c. of whole blood into the subcutaneous interscapular

tissues or into the buttocks. An all glass 10 or 15 c.c. syringe is boiled for five minutes, and half an ounce of liquid paraffin or alboline is boiled. Five grains of sodium citrate is boiled for five minutes in one ounce of saline. The syringe is filled with the hot liquid paraffin and expelled once or twice through the needle. Then the saline citrate is drawn up and expelled several times. The syringe is then ready for use and 10 c.c. of blood is withdrawn from the mother's vein and injected as above or into the superior longitudinal sinus. For infusion into the latter, 50 to 100 c.c. is sufficient, and if repeated, the former quantity is ample.

The Donor

The donor may be male or female, relative, friend, or stranger, free from disease and with a negative Wassermann. Preferably a donor from the same blood group as the recipient is desirable, but it is easier and quicker to have available tested donors who belong to group 4. They are the universal donors. In our cases we first tested the recipient against the proposed donor, searching till a suitable one was found, which often meant a large number of cross tests. More recently with the stock sera of groups 2 and 3 we have readily grouped the recipient and the donor, and in some cases have merely tested the available donors to locate one in group 4, and then carried out the transfusion. In four recent transfusions I have twice used the same donor. It is claimed that after a moderate transfusion a donor will quickly regain his usual blood count and also gain weight temporarily. Robust donors may be used eight times in twelve months.

In over fifty cases I have but once found that the donor has been even temporarily the worse for giving blood. In this case 850 c.c. was transfused at one time and the donor complained of lassitude for some weeks. Many donors have been medical students and have not experienced any inconvenience, and have been greatly interested in the whole procedure from the beginning of their donation to the completion of the recipient's intake.

When the patient's arm and leg veins are small and empty, the jugular veins may often be used.[20]

In only one instance have I found recorded as a result of blood transfusion *per se* a fatal result other than that due to hæmolysis. This occurred in a case reported by Goormaghtigh, where the recipient died as a result of arrest of the kidney function with anuria, and post-mortem findings of a grave toxæmia, with pathological changes in the liver and heart.

It would appear that to-day blood transfusion is a definite and valuable therapeutic asset in the treatment of anæmia of varying ætiology. No general practitioner need hesitate to employ it provided he has familiarized himself with the necessary technique for making the blood tests required, and has a glass graduate or two and the facilities for sterilizing the needed solutions for transfusion by the citrate method.

All medical schools should include in their course on hæmatology the laboratory tests for grouping bloods, and all final year students should have an opportunity of seeing or assisting at several transfusions. Preparedness in cases of hæmorrhage will, as in war, lead to victory.

SUMMARY OF OUR RECENT CITRATE CASES

Condition requiring Transfusion	Results	Reactions		
		None	Mild	Marked
Pernicious anæmia	Improved...10	6	1	3
Gastric or duodenal ulcer	Improved... 3	2	0	1
Preparatory for operation	Improved... 3	1	1	1
Post-hæmorrhagic anæmia	Improved... 1		1	
Secondary anæmia	Improved... 3	3		
Splenic anæmia	Improved... 1	1		
Hæmorrhage with jaundice	Improved... 1	1		
Parenchymatous nephritis	Not improved 1	Agglutination and hæmolysis		
Pulmonary tuberculosis	Improved... 2	2		
Pneumonia and influenza	Not improved 2	2		

Twenty-seven cases showed no reaction eighteen times; slight reactions, three times; marked reactions, five times, or 29·6 per cent.; agglutination and hæmolysis once.

BIBLIOGRAPHY

1. PEMBERTON, J. DE J.—Quoted in "Blood Transfusion". *Surgery, Gynecology and Obstetrics*, March, 1919, page 263.

2. LEE, ROBERT I.—"Field Observations on Blood Volume in Wound Hæmorrhage and Shock." *Amer. Jour. of Med. Sc.*, October, 1919, page 570.

3. CRABTREE, E. G.—"Blood Transfusion in War Surgery in the British Army." *Bost. Med. and Surg. Jour.*, 1919, clxxxi, 60.

4. CHARLES, R and SLADDEN, A. F.—"Resuscitation Work in a Casualty Clearing Station." *Brit. Med. Jour.*, 1919, i, 402.

5. BERNHEIM, B. M.—"Hæmorrhage and Blood Transfusion in the War." *Jour. Amer. Med. Assoc.*, 1919, lxxiii, 172.

6. BERNHEIM, B. M.—"Blood Transfusion, Hæmorrhage and the Anæmias." Page 50-51, Monograph publication.

7. LEE, ROBERT I.—"A Simple and Rapid Method of Selection of Donors for Transfusion." *Brit. Med. Jour.*, 1917, 2, 684.

8. HARTMAN, FRANK W.—"New Methods for Blood Transfusion and Serum Therapy." *Jour. Amer. Med. Assoc.*, November 16th, 1918.

9. DRINKER, C. K. and BRITTLINGHAM, H. H.—"The Cause of the Reactions Following Transfusion of Citrated Blood." *Arch. Int. Med.*, February 15th, 1919.

10. GARBAT, A. L.—"Soda Citrate Transfusions—100 cases." *Jour. Amer. Med. Assoc.*, 1919, lxxii.

11. ASHBY, W.—"The Determination of the Length of Life of Transfused Blood Corpuscles in Man." *Jour. Exper. Med.*, March, 1919, xxix, 267.

12. LEWISOHN, RICHARD.—"Clinical Results in 200 Transfusions of Citrated Blood." *Amer. Jour. Med. Scs.*, February, 1919, page 253.

13. *Jour. Amer. Med. Assoc.*, 1911, lvii, page 383.

14. FARR, R. E.—*Surg. Gyn and Obstet.*, March, 1919, page 327. "Transfusion Apparatus."

15. FLEMING A, and PORTEOUS, A. B.—*Lancet*, June 7th, 1919, page 973, cxcvi. "Blood Transfusion by Citrate Method—100 cases at a Base Hospital."

16. (a) TARR, T. S.—"Use of Superior Longitudinal Sinus in Infancy for Tests of Blood and Infusion." *Arch. Pediat.*, 1919, xxxvi, 72.

(b) GOLDBLOOM.—"A New Apparatus for Puncture of the Superior Longitudinal Sinus." *Amer. Jour. Diseases of Children*, vol. xvi, 1918. page 388.

(c) FISCHER, L.—*N. Y. Jour. of Med.*, 1919, xix, 183.

17. THOMPKINS, E. H.—"Basal Metabolism in Anæmia with especial reference to the effect of Blood Transfusion on the Metabolism in Pernicious Anæmia." *Arch. Int. Med.*, 1919, xxiii, 441.

18. LINDEMAN, E.—"214 Consecutive Cases without Chill by the Syringe Method." *Jour. Amer. Med. Assoc.*, 1919, lxvii, 1661.

19. HUNT, V. C.—"Reactions following Blood Transfusion by Citrate Method." *Texas State Jour. of Med.*, 1918, xiv, 192.

20. KALISKI, D. J.—"The use of the Superficial Jugular Veins for Infusions." *Jour. Amer. Med. Assoc.*, 1919, lxxii, 163.

21. HUCK, J. C.—"Changes in the Blood immediately following Blood Transfusion." *Bull. Johns Hopkins Hos.*, 1919, xxx, 63.

22. GOORMAGHTIGH, N.—"Arrest of Kidney Function following Blood Transfusion." *Arch. Med. Belges*, 1918, lxxii, 611. Abstract in *Surg. Gyn. and Obstet.*, July, 1919, page 793.

23. MCCLURE, R. D.—*Jour. Amer. Med. Assoc.*, 1916, page 793.

24. HOOKER, RANSOM.—*Arch. Int. Med.*, January, 1915.

HARRISON, BENJ. T.—"Blood Transfusion at the Front Area." *Jour. Amer. Med. Assoc.*, 1918, lxxi, page 1403.

Case Report

NOTES ON TWO CASES OF PECULIAR MEDICAL INTEREST

1. Syphilitic Aortic Insufficiency.
2. Cerebro-spinal Fever supervening upon (?) Erythema Nodosum.

By J. C. Meakins, *Lieutenant-Colonel, C.A.M.C.*

1. Staff-Sergeant-Major T. J. S.; Reg. No. T. 9063; A. S. C.; aged forty-seven.

This warrant officer was admitted to hospital on February 10th, 1918, with a diagnosis of influenza. He had served in the army since 1889, and his medical history sheet showed no illnesses of importance except malaria in 1901. He was married, having nine children, six alive and well.

Three days before admission he had a chill, followed by fever, headache, pains in the back, and a general feeling of weakness. On admission he had a temperature of 100°, respirations 18, and pulse 80. Physical examination showed no abnormality apart from the cardio-vascular system. There was conspicuous pulsation of the brachial and carotid arteries, and a distinct epigastric impulse; the pulse was regular between 70 and 80 per minute, and of a moderately collapsing character. The blood-pressure was 150. Heart: Apex beat diffuse, point of maximum impulse in the fifth interspace at 10 cm. to left of mid-sternal line. The cardiac dulness extended 10 cm. to left and 4 cm. to right. On auscultation the sounds over the pulmonary area were clear. The second sound over the aortic area was of very poor quality, and was followed by a long blowing diastolic murmur, which was transmitted down the sternum and towards the apex region, where it was less distinctly heard, and was associated with a faint systolic murmur. *X*-ray examination showed very little dilatation of the aorta; heart shadow not conspicuously enlarged.

From the Medical Division of No. 15 Canadian General (Duchess of Connaught, Canadian Red Cross) Hospital, Taplow, Bucks.

On February 13th, 1918, Wassermann reaction was positive. On February 14th, 1918, potassium iodide and mercurial inunctions commenced. On March 7th, 1918, Wassermann still positive, but the diastolic murmur, although still present, was much less distinct. The potassium iodide and mercury treatment was continued, and during April he received 1·4 grm. of galyl intravenously, divided into five doses at six-day intervals. On April 30th, 1918, the diastolic murmur had completely disappeared, and the systolic murmur at the apex was very faint, and during the ensuing week disappeared also. On May 8th, 1918, the Wassermann was still positive, but all evidence of cardiac lesion had disappeared.

The interesting point in this case is the recovery, while under treatment, of a well-marked aortic insufficiency of syphilitic origin.

2. Driver H. H. N., Reg. No. 30622; A.F.A.; age nineteen.

This private reported sick in France on April 12th, 1918, complaining of headache, pain in the shins and knees, swelling over the shins, and fever. He was sent to a detention hospital, where he remained for two weeks. During this time bright red circinate patches developed on the shins, forearms and thighs. He was transferred to hospital, and evacuated to England on May 9th, 1918, with a diagnosis of erythema nodosum.

On admission to this hospital he had pains in the small joints of the hands, in the knees and ankles, and the erythematous patches were still present on the forearms, and gave all the appearance of typical erythema nodosum. Temperature ranged from 101° to 103° F. There was pain in the joints, with some swelling of the knees and ankles, and redness about several joints in the hands, particularly the left metacarpal phalangeal joints. Heart and other systems normal. On May 10th, 1918, blood count: Polymorphs, 68 per cent.; large mononuclears, 16 per cent.; small mononuclears, 16 per cent.; red cells, 4,900,000; white cells, 8,700; hæmoglobin, 95 per cent. On May 15th, 1918, blood culture sterile. On May 16th, 1918, white cells 20,600. On May 20th, 1918, blood culture sterile. During this period temperature was intermittent; he had irregular and recurrent swellings of various joints, profuse sweats, and the areas of erythema developed over the legs and arms at various intervals. On the morning of May 22nd, 1918, he had a sudden onset of severe headache, vomiting and restlessness. White blood cells, 14,500; polymorphs, 80 per cent.; large mononuclears, 9 per cent.; small mononuclears, 11 per cent. There was slight retraction of the head and considerable photophobia. Lumbar puncture showed turbid fluid, and 30 c.c. of anti-meningococcal

serum were injected intraspinally. Examination of fundi was normal. During the day retraction of the head became more pronounced, and Kernig's sign distinctly positive. Examination of spinal fluid, cell count 6,000 per c.mm., cells being chiefly polymorphonuclear. No organisms found in smears. Examination of the eyes on May 23rd, 1918, showed the veins engorged; otherwise normal. Lumbar puncture on same date showed slight increase of pressure and turbidity; temperature was lower, and the patient felt somewhat better. On May 24th, 1918, he still complained of headache, there was slight retraction of the neck, and Kernig's sign was doubtful. Temperature was normal; lumbar puncture revealed clear fluid under no pressure. The culture of the original spinal fluid showed a very slight growth, which on agglutination was determined to be meningococcus, Type 3. Patient made an uninterrupted recovery from the meningitis, although the arthritic symptoms and the erythema persisted for many weeks. Repeated cultures from the naso-pharynx, both during the acute stage and the convalescence of the meningitis, failed to show any meningococci.

After recovery from the meningitis, the following additional history was obtained from the patient: Left Australia on November 23rd, 1916, and on the transport, three cases of meningitis developed, with two deaths. As far as he knows, he did not come into contact with these cases. On December 26th, 1917, patient spent leave with his brother, a close associate of whom died of meningitis about this date.

This case was of particular interest in that epidemic cerebrospinal meningitis developed in a patient already suffering from a febrile illness accompanied by a pronounced rash.

An address of great and immediate public interest was given, at a meeting of the British Association for the Advancement of Science, by Dr. W. Brown, on hypnotism and mental analysis as applied to soldiers suffering from shell shock, gun shock, and other maladies. In 1916, Dr. Brown was appointed neurologist to one of the armies in France, and while engaged in this service, he became more and more convinced of the importance of hypnotism in nervous cases of this character. Many of the men had been, as it were, hypnotized by the sheer shock of the explosion itself, and he found that by inducing an hypnotic sleep influences could be exerted which in many cases were effective for good.

Editorial

THE DEATH OF SIR WILLIAM OSLER

THIS issue of the JOURNAL opens with the life-story of one whose name is as a household word among us, whose scientific enthusiasm has leavened and clinical acumen inspired the medical teaching of the past three generations, and whose warm hearted hospitality has been shared by the profession of two continents these many years. Those portals now are darkened, and the light of mingled human kindness and genius that shone from those burning eyes has been forever quenched. To us there remains a great sadness, and an inheritance that is inviolate,—the name of the great Canadian physician, and the tradition of his early formative years. It is proposed to publish shortly, for the benefit of all our subscribers, a special memorial number of the JOURNAL which will be additional to the regular monthly issues, and which will be dedicated to his memory.

ENCEPHALITIS LETHARGICA

UNDER this title a number of cases have been reported from Europe and on this continent within the past two years. The earliest cases were described by von Economo in Vienna in 1917. Occurring in epidemic form, the most prominent feature was a prolonged somnolence ending either in recovery, or after weeks or even months of lethargy, in death. The onset was marked by headache, vomiting, and fever, whilst cranial nerve paralyses were frequently but not always present. Von Economo regarded the condition as a specific infection allied to, but not identical with, the cerebral type of poliomyelitis.

The occurrence of cases in Canada and in the United States has awakened considerable interest throughout the country, and we publish in this number an important con-

tribution to our knowledge of the clinical and pathological features of this disease as met with in its recent outbreak in Winnipeg.

The morbid changes, while in some instances invisible to the naked eye, in others appear as small hæmorrhages in the brain stem and basal ganglia; the former may present evidences of their existence during life by an involvement of the cranial nerves; the latter by a syndrome resembling the symptoms in Parkinson's disease.

Examples of cortical and subcortical involvement are recorded in Boyd's paper; Bastien has also reported a series of cases with cortical lesions and symptoms. Considerable cortical hæmorrhage may occur, giving rise to irritative symptoms, hemiplegia, or even apoplexy. When such hæmorhage exists, this lesion may be regarded as primary, and the inflammatory changes leading to it may be easily overlooked. Visible thrombosis of surface veins may, however, give a clue to the nature of the malady, and indicate its inflammatory character.

Although the examination of the spinal fluid may prove negative, in a considerable number of cases a mononuclear increase of cells varying from slightly above the normal to 200 per cmm. has occurred, sometimes with a globulin reaction. In such instances meningeal involvement may be anticipated and the signs of meningitis, such as rigidity of the neck and limbs and the Kernig sign may be present.

Opinions are divided on the relationship of encephalitis and poliomyelitis. English and French observers mostly regard these diseases as distinct entities. Epidemiological evidence collected by James (*Lancet*, December 21st, 1919) is in favour of this view. Typical spinal poliomyelitis might be expected in districts where the disease was prevalent, if both were due to the same organism, and yet in fifty-one sanitary districts from which the new syndrome was reported, only twenty-six also recorded cases of poliomyelitis; the remaining twenty-five had no instances of this ailment.

In favour of the identity of the two maladies may be urged the facts that the morbid changes and clinical symptoms are very similar. The experiments of Breinl in Australia on encephalitis and of Flexner on poliomyelitis are also in remarkable agreement. Both observers were able to transmit the disease to monkeys with the filtered virus and from these cases other monkeys were successfully inoculated. In Flexner's cases, however, the spinal cord was attacked whilst in Breinl's the brain as well was involved in the morbid process.

Poliomyelitis as an acute specific disease is generally regarded as producing immunity from subsequent attacks, but in one of Boyd's cases, encephalitis occurred in an individual who had passed through a previous attack of poliomyelitis, a fact strongly opposed to the identity of the two maladies. That they may, however, be closely allied is supported by the analogy of typhoid and the paratyphoid infections. Immunity is not conferred by typhoid for the paratyphoid infections and conversely, and yet the distinction between these affections is only possible by bacteriological reactions. Until the ætiological factor of both poliomyelitis and encephalitis is established, the matter must remain a subject of speculation.

The relationship of encephalitis to influenza has evoked a good deal of discussion. The occurrence of sleeping sickness following influenza has been noted for over two hundred years and has also been recorded in recent epidemics. The cases were marked by stupor and somnolence with ophthalmoplegia, and have been described under the name of "Nona", probably a corruption of the word coma. That cases should follow influenza is to be expected owing to the widespread character of this malady. Numerous instances, however, have been recorded in which there was no preceding influenza, so that the connection is not a specific one, and there is not even proof to show that the lessened resisting power, induced by an attack of influenza, renders its victims more susceptible to the infection of encephalitis.

Section of Therapeutics

SYSTEM IN THE CARE OF THE SICK

By Robert Dawson Rudolf, C.B.E., M.D. (Edin.), F.R.C.P. (Lond.)

Professor of Therapeutics in the University of Toronto

WHEN a person suffering from some disability sends for a physician, he does so chiefly in the hope that he may be cured, or at least relieved from his distress and incapacity.

Now, in the physical examination of a patient, it has always been recognized that the best results are obtained if the examiner uses a certain routine in his work, and the one usually taken is that of inspection, palpation, percussion and, if necessary, auscultation. In exactly the same way, when the practitioner comes to the consideration of the treatment of the case he will find it useful to follow a certain routine. The one adopted here, and which I have always found of most value, is the following:

1. Diagnosis.
2. Environment.
3. Diet.
4. Removal of the cause of the ailment (specific therapy).
5. Symptomatic treatment.

By using some such method, the physician's directions will be given in an orderly manner and the tendency, all too prevalent, of immediately prescribing some medicine and often leaving the patient without further detailed instructions, will be avoided. There are few places in the field of medicine where system is more required than in treatment and few where it is more often absent.

1. *Diagnosis.* The first duty of the practitioner is naturally to try and find out what his patient is suffering from, and he must, of course, leave no stone unturned with this object in view.

Some diagnoses are apparently complete, for example, one of erysipelas, and yet even such an apparently complete diagnosis is by no means sufficient for the therapeutist. Thus, the patient may also have nephritis, due to the toxins of the infection or owing to

some previous condition. The diagnosis of a case includes not only the recognition of some disease, but also the whole condition of the sufferer, the state of his resisting power, etc. Two patients are suffering from, let us say, erysipelas, the one a previously healthy young adult and the other a broken-down alcoholic, with doubtful kidneys and thickened vessels. Our treatment must be quite different in the two cases, although they are both examples of erysipelas.

Some disorders are definite entities, but the young practitioner will soon find that most of his work will consist in the care of people suffering from slight disturbances of the various functions of the body to which it is hard to give a name. Yet these slight conditions are often very disabling and require treatment. It is usually easy to treat a condition early and difficult to do so when the disease has become fully established. It is early in the treatment of disease, as in the treatment of bad habits, that most may be done.

Many diagnoses pass through the three stages of possibility, probability and then certainty. For example, a patient may have fever, headache and some indefinite pains over the body. At the first visit a large number of possibilities will occur in the mind of the physician. His patient may have typhoid or paratyphoid, he may have influenza, or cerebro-spinal meningitis, or miliary tuberculosis, and so on. In a day or two the diagnosis will have been fined down to the probabilities of say typhoid or one of the paratyphoid infections, and a little later it may be quite clear that the disease is, say, typhoid.

Only too often, unfortunately, the diagnosis is uncertain until the condition ends in death or terminates in recovery.

Now it is most important that during these days of uncertainty and delay something be done for the patient; symptomatic treatment to be sure, but none the less necessary for the relief of the suffering and distress.

Even when a diagnosis appears, according to our lights, to be clear, further pathological work in years to come may show that it was wrong. The history of medicine is full of such examples. And yet how often has the treatment been superior to the diagnosis! For ages, malaria was believed to be due to an evil spirit, which, entering the body of the patient, gave rise to the fever and other distress, and yet the administration of quinine, or rather, cinchona bark, although used as a spirit scarer, was excellent. After the plasmodium of malaria was discovered, it was believed that the quinine directly destroyed this. The latest belief is that the drug

does not directly kill the parasite, but stimulates the tissues to produce an antibody which does so. But all along, quinine or cinchona have been given for the relief of malarial-stricken patients.

A diagnosis may be right and the treatment wrong, as judged by the accepted treatment of the day, and yet the patient may recover. The patient thus often gets better in spite of treatment, so strong in many cases is the natural tendency towards a return to the state of health. This *vis medicatrix naturæ* has always been the doctor's best friend. In most cases all that the practitioner can do is to place his patient in the best possible surroundings, feed him wisely and relieve the symptoms as best he can, and then allow this great natural force to act.

The wise physician works along with and makes use of the natural curative forces, the less wise one thinks that he is curing the patient, and often in his attempts to do this, combats the *vis medicatrix naturæ*, often with dire results to the patient.

When a man has swallowed some corrosive poison, vomiting quickly occurs with the result that the poison may be ejected. The sound practitioner will encourage this natural attempt to get rid of the irritant, or at least will not interfere with it, the foolish one will give sedatives to relieve the vomiting and pain, and if he succeeds, the poison will remain and will work further mischief. Quite as definite examples of this tendency to interfere with nature's endeavours are or rather were rampant in the practice of medicine. One such may be mentioned. A fevered patient longs for water and it seems evident now that this craving is a natural one and that the water will help him to get rid of his fever —by increasing the sweat, inducing diuresis and so on. And yet for long periods in the history of medicine, under the sway of prevailing theory, water was forbidden to the fevered, much to the distress and suffering of the patients. Hence, when we are considering how we can best help a sick man, let us always first think well of the natural history of the condition and to what extent and how the *vis medicatrix naturæ* will act and then let our therapy all be so as to encourage those endeavours.

But it is possible to go too far in the direction of leaving the case to nature—in other words, so-called expectant treatment. It has been well said that nature is careful of the race and careless of the individual. When a man carries a tape-worm, does nature help him to get rid of it? In the same way, what tendency to cure is there in malignancy? Very frequently after the physician has fully studied the natural tendency to a return to health, he will

find that much remains for him to do if his patient is to reach that goal.

2. *Environment.* As soon as the practitioner has examined his patient, it becomes necessary to arrange for his environment. May he be allowed to go on with his work or must he lie up? Does he require skilled nursing? Should he be isolated? Such are a few points that call for settlement as regards the patient's surroundings.

Personality of the Physician. The bearing of the medical attendant has a very powerful effect for good or for evil upon the patient. Many a successful medical man owes his success more to his manner and way of dealing with his patients than to any scientific knowledge that he possesses. Especially is this important when we have to deal with so-called neurotic patients, but the most stoical person is much influenced for better or for worse by the physician's manner and style of dealing with him. "What doctor possesses such curative resources as are latent in a spark of happiness or a single ray of hope?" pathetically asked that charming but neurotic French writer, Amiel. But surely it is the privilege and the duty of the doctor to give such sparks of happiness and rays of hope, and if he cannot do so, then he has mistaken his profession, no matter how skilled he may be in all the medical sciences. A few cheerful words, a hopeful view of the case expressed in the hearing of the patient or his friends, will often do more good than all the drugs that may be prescribed.

Even in such definitely organic diseases as cancer of the stomach, one may see the cheerful bearing of a new practitioner called in to help cause a great improvement in the sufferer, even to the extent of causing him to put on considerable weight. Osler mentions such a case in his work (with T. Macrae) on cancer of the stomach, and I have had a similar experience.

The patient must have confidence in his doctor in order that treatment may be fully effectual, and if the doctor feels, as every practitioner occasionally does, that this confidence is missing, then it is far better for the patient, and for the practitioner in the long run, that a change should be made. So much depends upon the hope inspired by confidence in the practitioner and his remedies.

All that a practitioner learns in his intercourse with his patients must of course be kept secret. The question often arises, however, as to how much should be told to the patient himself or to his near relatives. Generally speaking, a sick man has a right to know about his condition, but often the medical attendant will see that to tell him the unvarnished truth will only do him harm. In the

first place, as has already been said, frequently the doctor does not know what is the matter, although he may suspect a number of possibilities, and it would never do to immediately tell the patient or even his near relatives all these suspicions.

Often the patient will worry his doctor for a name for his complaint and the practitioner can frequently satisfy this desire without unduly committing himself. Professor Chiene, of Edinburgh, used to tell his students that he had often given a patient comfort by telling him that he had a sacro-iliac synchondrosis and Sir William Gull tells of what great satisfaction the friends of a man suffering from some obscure disease had received when he assured them that the patient had a cachexia! In military practice one would often see a soldier quite proud of such a diagnosis as "P.U.O." (pyrexia of unknown origin) or even of "N.A.D." which means "no appreciable disease".

When a patient is seriously ill and the practitioner does not feel that he should know then it is always well to inform some near relative of the sick man of the situation, both in order that the physician may protect himself and also in order that the friends may prepare for the worst. It is sometimes the duty of the doctor to tell his patient that he should make his will, and if he is met by the anxious question:—"Then doctor, am I going to die?" he can always give the old reply, "It is surely better to live prepared than to die unprepared. A man never yet shortened his life by making his will."

When the worst seems inevitable, the practitioner should still be very chary about giving up all hope. Such an action will almost certainly condemn the patient, already very ill, to a quick death. We are all liable to make mistakes, and it is not a rare thing to meet people who at least say that they had been given up by their doctor and who are still alive and well years after. I can recall the case of an old lady who had been condemned to death by her doctor during a severe attack of pneumonia. In spite of this gloomy prognosis she recovered. The doctor then announced that at any rate she could not live through another winter. That was some years ago and the old lady is still going strong, and another practitioner attends her.

Even when the end is very near, the practitioner should not commit himself to days or hours. "Will he live until next Sunday, doctor, as our son is in the West, and would come home if he thought that he could see him alive?" "Will he live until daylight?" Such are the sort of questions that are asked when the friends hear

that the patient is dying. And to all such enquiries the practitioner will do well to give very guarded replies. A good way and the true way to answer is to say that it all depends upon the resisting powers of the sick man. We may add that *probably* he will live until next Sunday or until daylight as the case may be, but we have left ourselves a loophole and, after all, any more definite statement is mere guessing.

3. *Diet.* A certain amount of food is of course necessary to meet the caloric needs of the patient, although if he be in bed then less nourishment is required than when he is up and about.

The body has always a reserve and can stand a certain amount of starvation for a short time without serious damage. Occasionally it becomes necessary to stop all food for a day or two, but so-called "starvation cures" for fevers of long duration and for other prolonged conditions should always be looked upon with suspicion. It was the great Irish physician, Graves, who asked that the epitaph upon his tomb might be that "He fed fevers". I remember once in India seeing a poor native woman who had been in labour for eight days and during all that time she had been starved. It seemed that it was a Mahomedan custom that women should not eat while in child-bed.

4. *Specific Treatment.* (Removal of the cause.) It is often a simple matter to remove the cause of some bodily trouble and then the patient will quickly recover. Such specific therapy is seen when we empty the stomach of a poison or by giving an anthelmintic, rid him of a tape worm that may have caused ill-health for years. Again, in the administration of quinine in malaria, antitoxin in diphtheria and mercury in syphilis we are more or less directly attacking the cause of the trouble. Of a less direct but still similar nature is the use of vaccines, where we stimulate the patient's tissues to an even greater effort in the production of antibodies. Specific therapy is the ideal one and the number of diseased conditions that may thus be combatted is constantly growing, but it is still a fact that the vast majority of human ills are yet beyond such satisfactory methods of attack.

5. *Symptomatic Treatment.* Here we endeavour to lessen the sufferings of the patient and to combat symptoms that may in themselves threaten life or health, although we chiefly in the meantime rely upon the *vis medicatrix naturæ* to restore the patient to his normal. Yet, very often, by keeping up the strength of the patient by appropriate feeding or the use of stimulants, by saving him from the wearing effects of pain or of sleeplessness we can so

preserve his strength that the natural forces may have time to act and he may recover when otherwise he would have died.

The physiologist, Haldane, in writing of the administration of oxygen in inflammatory conditions of the lungs, says:—"It may be argued that such measures as the administration of oxygen are at the best only palliative and of no use, since they do not remove the cause of the pathological conditions. As a physiologist, I cannot for a moment agree with this reasoning. The living body is no machine, but constantly tending to maintain or to revert to the normal, and the respite afforded by such measures as the temporary administration of oxygen is not wasted, but utilized for recuperation." (*Brit. Med. Jour.*, Feb. 10th, 1919.) A symptom is usually a link in a vicious circle and if we can break that link then the patient may recover. A man has an eczema and suffers from pruritus which causes him to scratch, and the scratching increases the eczema. If the itchiness be lessened by appropriate sedatives, local or general, he will cease to scratch and the eczema will have a chance to get well. I always remember a good example of this breaking of a vicious circle that occurred in the case of a pet dog of mine many years ago. The animal had contracted some irritative skin lesion and spent his time in scratching, and soon his skin was raw and his whole state so pitiable that I decided to put him out of his agony. I gave him an enormous dose of opium and left him to die in an outhouse. Next morning he was not dead, but soundly asleep, and he continued in this peaceful state for two or three days, and when he came to himself his skin had so far healed that he did not scratch again and soon was quite well. In cases like this, and one might multiply them indefinitely, the treating of symptoms brings about recovery when otherwise this might not occur.

One sees the curative effect of symptomatic therapy when a surgeon sets a broken limb. His splints merely lessen the abnormal mobility of the broken fragments of bone and thus lessen the pain and then the natural healing forces have a chance to act. "I dressed his wounds. God healed them," was the modest and true reply of the great Ambrose Paré when he was congratulated upon having cured some patient. In the same way, when a physician gives bromides to an irritable and sleepless neurasthenic, he, so to speak, splints the jaded nerves and thus gives nature the chance to act.

In symptomatic treatment drugs are chiefly used. It has often been said of drugs that they sometimes cure, often relieve,

and always console, and there is much truth in the remark. What comfort, which cannot be explained pharmacologically, is often given by a bottle of medicine, and surely if the physician sees that he can give such comfort it is his duty to do so. I have never yet prescribed a bread pill or a bottle of coloured water, but one can easily see that such a pill or draught given with assurance and taken in faith might do good when such a result could not be attained in the absence of all medicinal treatment. It is a mistake to leave a patient for days without any treatment just because the diagnosis is not yet clear or indeed because it is only too clear and believedly cannot be cured by treatment. Remember, we are dealing with human beings who are sick, rather than treating diseases. Moreover, there is always some symptom, such as sleeplessness, headache, constipation, want of appetite, and what not, which can be influenced, even if we do not yet know the full diagnosis nor can do much for the underlying disease. In hospital practice especially, too much time is apt to be spent over the pathological aspect of the case and too little over the question of what can we do for the sufferer.

But there is good and bad symptomatic treatment. As an example of the former, one might mention the free relief of cough and pain in dry pleurisy by the use of opiates. Bad symptomatic therapy is seen when opiates are freely given to relieve a cough where there is much bronchial secretion. The patient's very life depends upon his coughing up this material and the opiate will lessen this cough and will also tend to weaken the respiratory centre.

Often, again, it is important not to mask symptoms until a diagnosis has been arrived at. This applies chiefly to acute abdominal conditions where operation may be required. But, all the same, when it has been decided to operate, in any hours that may elapse between this decision and the operation, the pain and distress should be freely lessened by anodynes. This is sometimes forgotten and such an omission may be the cause of much unnecessary and damaging suffering.

Again, in inoperable cases of cancer, where the patient's days are numbered, it is surely the physician's duty to use sedatives freely. Here there is no question of habit, and often it appears that life can not only be made comparatively comfortable, but may actually be prolonged by such merciful therapy.

Thus, to summarize, it is urged that the clinician should be just as systematic in his treatment of the sick man as he probably is in his physical examination of him.

Correspondence

ON THE PREVALENCE OF VENEREAL DISEASE IN CANADA

To the Editor:

In your September issue, a letter from Professor A. B. Maccallum was printed concerning the prevalence of venereal diseases in Canada. Professor Macallum seems to think that there is comparatively little syphilis in the country, but his letter showed so plainly that existing statistics had not been consulted, and he seemed to me to be so obviously in the wrong that I hesitated to comment regarding his statement. Since I was the original author of the remark to which he particularly objects, however, perhaps some statement by me will not be out of order.

The statement as originally made at the Ottawa conference was as follows: "It is stated by the New York Department of Health that 8 per cent. of the population of this continent have syphilis. If this is true, there are over half a million cases of syphilis in Canada." As a matter of fact, 500,000 cases means only about 6 per cent. of the population, and it would seem in the light of available evidence that this estimate is fairly close to the truth.

The Royal Commission in Great Britain came to the conclusion that "the number of persons who have been infected with syphilis, acquired and congenital, cannot fall below 10 per cent. of the whole population in large cities and the percentage affected with gonorrhœa must greatly exceed this proportion". Dr. Douglas White who has given special attention to the statistical side of venereal disease has pointed out that this probably means 450,000 syphilitics in London alone and that there are likely about three million syphilitics in the United Kingdom. Sir Malcolm Morris in quoting the above statements estimates that cases of gonorrhœa are probably several times as numerous as those of syphilis.

The estimates by Erb that 12 per cent. of the population of Berlin is infected with syphilis and by Fournier that 15 per cent. of the population of Paris is infected are well known.

Hospital statistics although not an absolute criterion of the amount of syphilis among well persons, would seen to indicate an

appalling amount of syphilis throughout the country. In the United States a series of routine Wassermann reactions on white patients done in a number of hospitals showed an average positive percentage of 19·6. These ranged from the 28·1 per cent. in the Post-Graduate, New York, and 25·7 per cent. in Bellevue down to 12·7 per cent. in St. Luke's, San Francisco. In all, 18,187 patients were examined. In Canada, we have the statement that in a period of three months in 1917 routine Wassermanns in Toronto General Hospital showed a positive percentage of 12, while a similar survey made in Montreal General Hospital showed a percentage of 26. These I understand did not include cases specifically admitted for venereal disease. In an average year, about 25 per cent. of the admissions to Toronto Hospital for the Insane consist of general paresis.

A great deal of information as to the prevalence of venereal disease was gathered in the army—quite sufficient to inform us not only that venereal diseases are very prevalent, but also that conditions in civil life are worse than in the army. The statement of American authorities that in five sixths of all cases discovered among men in uniform, the disease had been contracted previous to enlistment is illuminating. In one draftee regiment in Toronto, we found 5·7 per cent. of the men to have syphilis, by means of the routine Wassermann. These men were examined within a few weeks after enlistment and were all A2 men supposedly absolutely free from all infection.

I need not dwell on the end results of venereal diseases here. Every physician knows how serious they are. I trust, however, that this brief résumé of a few of the known facts as to their prevalence will be of value to your readers.

As to just why Professor Macallum should find it necessary to speak of *wild* estimates as to the prevalence of these diseases I do not know. Possibly, however, the above will prove of value to him and perhaps make him feel with all social hygiene workers that in venereal diseases we are confronted with a problem which is greater in its magnitude and in its possibilities than any public health problem we have approached in the past.

Yours sincerely,

GORDON BATES,

General Secretary.

To the Editor:

Dr. Bates appears to think that because the New York Department of Public Health has stated that 8 per cent. of the population of this continent (United States) is afflicted with syphilis, he is justified in putting the number of syphilitics in Caanda at half a million at least.

The New York Department of Public Health is not in a position to justify such an estimate. It has not the statistics for the continent, because they do not exist. There is no organization which has systematically attempted to gather such statistics. Doubtless the department, in making such an estimate, must have based it on statistics gathered from hospitals in New York and elsewhere, the patients of which hail chiefly from slum districts, where syphilis flourishes as it does also in the slum districts of Old World cities. It would be as absurd to accept such data whereon to base an estimate of the prevalence of syphilis in the United States as it would be to conclude that there are over 150,000 syphilitics in Montreal because the application of the Wassermann showed that 26 per cent. of the patients in the Montreal General Hospital had the disease!

The prevalence of gonorrhœa is sometimes estimated to be as high as six, but not lower than three, times that of syphilis. According to the Royal Commission on Syphilis, the cases of gonorrhœa must greatly exceed those of syphilis in large cities, and Sir Malcolm Morris seems to accept this proportion. Dr. McPhedran, in the November issue of the Journal, quoted the statistics for the two diseases for the State of Victoria, Australia, for 1918, in which the number of cases of gonorrhœa is to those of syphilis as 3 to 1. Apply this finding to the estimate of the New York Department of Public Health, and there results 24 per cent. as the proportion of the population of the United States afflicted with gonorrhœa, or 32 per cent. for both diseases!

Does anyone seriously believe that more than 30,000,000 of the population of the United States have venereal disease, or, approximately, one out of every three persons? Can anyone seriously contend that because 12 per cent. of the patients in the Toronto General Hospital are syphilitics, nearly 4,000,000 or half of the population in Canada are afflicted with the one or the other disease? Is Dr. Bates, who estimates that there are at least half a million syphilitics in Canada, prepared to maintain that in the Dominion, there are now 2,000,000 venereal cases, or one out of every four of the population?

More than twenty years ago I endeavoured to get the data for an historical study of syphilis, but had to desist because I found the estimates of its prevalence in the past, then obtainable, to be utterly unreliable. It would seem, to put it mildly, that there is not much improvement in this respect as regards the estimates of its prevalence to-day. The coldly critical faculty appears to be lacking, either wholly or partly, in a certain class of syphilologists.

I must again earnestly protest against the publication of exaggerated estimates of the prevalence of syphilis in Canada, especially in the public press, and, more especially, when disseminated, as Dr. Bates' estimate was, by the Associated Press. These estimates do incalculable harm to the medical profession and to the cause in whose interest they are advanced. Physicians and others in my circle of acquaintance, in referring in conversation to the estimate of half a million cases of syphilis in Canada, have expressed themselves as extremely sceptical regarding it. One gentleman, a bank manager, even claimed that "the doctors are attempting to put one over the public" regarding syphilis. All these, in consequence, naturally tend to minimize the seriousness of the prevalence of the disease.

Even the plea, if advanced, that "the end justified the means", in this matter is wholly uncalled for. Imagine the fifty thousand syphilitics in Canada, if there are so many, all gathered by themselves in a city. All the governmental machinery possible would be employed to quarantine it and all the methods of treatment available would be used to stamp out the disease. Is not the seriousness of its prevalence enormously enhanced by the distribution of that number throughout Canada, infecting others and increasing the incidence of the disease in our people?

A. B. MACALLUM

OTTAWA, December 12th, 1919.

[With the publication of these two letters, lack of space compels us to close our pages to further correspondence on this subject. In doing so, however, we may quote the following written with reference to similar statistics, and clipped from the editorial columns of the Journal of the *American Medical Journal*, December 13th, 1919:

"As the existence of a previous syphilitic infection predisposes to other diseases as well as sends many patients to the hospital with the late manifestations of syphilis in the internal organs, the figures cannot be considered applicable to the general population."—EDITORS.]

GREAT VALUE OF SERVICES RENDERED BY LIEUTENANT-COLONEL R. P. CAMPBELL AND COLONEL ROBERT WILSON

To the Editor:

On returning from overseas very recently, while reading the December number of the Canadian Medical Association Journal, I learned with great regret of the death of Robert Wilson. Memories of September, 1914, are recalled and of friendships which could only be interrupted by death itself. Of my comrades in the same General Hospital, at that date, two are now gone, both very able and valued men. I had got to know and appreciate them with an affection which I think was mutual.

When first I met R. P. Campbell some fifteen years ago, he was working with Von Mikulicz at Breslau; later on, as a comrade, I fully realized how it was that his Montreal colleagues had held him in such high esteem. His great ability, his splendid scientific mind, his quiet effective work carried on in the most unassuming way could not but impress one. Killed in action, when in command of a field ambulance in 1916, lost to Canada a fine soldier, a highly accomplished surgeon and a gentleman.

Robert Wilson was a man full of energy with great capacity for work, impulsive and warm hearted to a degree. His splendid technical knowledge of *x*-ray and electro-therapeutics was by no means the limit of his accomplishments. He had a grasp of hydrotherapy, physical exercise and massage not commonly possessed by medical men, which made his work of enormous value to the Canadian medical service. The urgent need of special means of treatment for disabilities soon became apparent in the war and much credit is due to Robert Wilson for his share in placing the Canadian medical service in the forefront at an early date as regards the special methods which were rapidly adopted in many orthopædic centres. It is a matter of pride that the first functional training in Britain in military hospitals was given in the Granville Canadian Special Hospital, Ramsgate. Here at that time Wilson worked and instituted the early classes of instruction in massage and physical exercise.

And so I now wish to make reference to these two medical officers who have done their work so nobly and well, not in any

complete and descriptive sense, but in grateful recollection and to place a little sprig of myrtle to their memory.

MURRAY MACLAREN.

ST. JOHN, N.B., December 15th, 1919.

Retrospect

ŒDEMAS, NOT OF NEPHRITIC ORIGIN, IN INFANCY AND CHILDHOOD

BY ALTON GOLDBLOOM, M.D.
Montreal

MORSE, J. LOVETT: "THREE UNUSUAL CASES OF ANGINONEUROTIC ŒDEMA IN INFANCY." *Boston Med. and Surg. Jour.*, vol. cxlii, 1900, page 10.

FAIRBANKS, ARTHUR W.: "THE 'IDIOPATHIC' OR 'ESSENTIAL' DROPSIES OF CHILDHOOD." *Am. Jour. Med. Sci.*, September, 1903, page 443.

FLEISCHMANN AND WOLFF: "ANGEBORENE WASSERSUCHT." *Arch. f. Kinderh*, vol. lxii, 1914, page 75.

HOLT, COURTENEY AND FALES. "CHEMICAL STUDY OF WOMEN'S MILK, ESPECIALLY ITS INORGANIC CONSTITUENTS." *Am. Jour. Dis. Child*, vol. x, 1915, page 229.

COURTENEY AND FALES: "STUDIES IN INFANT METABOLISM, NUTRITION, COMPOSITION AND PREPARATION OF PROTEIN MILK." *Am. Jour. Dis. Child*, vol. x, 1915, page 172.

COURTENEY AND FALES: "BLOOD SOLIDS." *Am. Jour. Dis. Child*, vol. xiv, page 202.

THERE is perhaps no more interesting problem in pediatrics than that presented by the œdemas which occur in various conditions in infancy and childhood. There is a tendency to ascribe all dropsies in children to nephritis. This is perhaps due to the fact that the occurrences of other types of œdema has never been sufficiently em.hasized in the literature. There is, however, a large group of œdemas, particularly those which are seen in young infants, in which no renal or cardiac lesions have ever been demonstrated. Very little indeed, is known of the pathogenesis of these œdemas any more than that all of them are associated with exces-

sive salt retention in the tissues, such as sometimes occurs, for instance, when sodium chloride is added to dilutions of cow's milk already rich in salt. Although salt retention accompanies every form of general œdema, it is not known whether this is secondary to water retention or vice versa. Of the factors which determine the salt balance in nutritional disturbances of children, there is also very little known. The relationship between salt retention and water retention can readily be demonstrated. Bauer has studied the sodium chloride metabolism in artificially fed infants suffering from nutritional disturbances. He has shown that the weight can be varied at will by feeding either salt rich or salt poor formulæ. The increase in weight on a salt rich diet is not due to differences in tissue building, but to differences in the amount of water retained by the blood and tissues. Extreme degrees of water retention will give rise to a generalized œdema which is not due to any kidney lesion.

Babies who are fed on dilutions of cow's milk must necessarily excrete a greater quantity of salts than babies who are fed on the breast. A litre of cow's milk contains about 7 gm. of total salts, while a litre of woman's milk contains only about 2 gm. It is evident, therefore, that even in formulæ containing equal parts of cow's milk and diluent, the total salt content is nearly twice that of undiluted breast milk. This means that in all the ordinary cow's milk dilutions the sodium chloride intake will be nearly doubled. The proportion of sodium chloride in the total salts of breast milk and of cow's milk is almost the same. A thriving baby who is artificially fed, will overcome this excessive intake by increased chloride excretion in the urnie. A baby suffering from nutritional disturbance has an unstable salt equilibrium.

According to Michel, quoted by Bauer, the daily salt retention in a normal nursing infant, whose intake was ·22 gm. of total salts per day, was ·095 gm. and his excretion in the urine and fæces ·124 gm. In an artificially fed infant, who was not gaining, and who was fed undiluted cow's milk, the intake was 1 gm. per day, the excretion ·876 gm. and the retention .219 gm.; nearly twice that of the breast fed baby. These two infants, though not quite the same age, were about the same weight. The occurrence of œdema in a child of the latter type might be explained on the basis of a derangement in the excretory powers of tissues overtaxed by an excessive salt metabolism.

These nutritional œdemas, as they are called, do not occur in healthy, thriving infants, but rather in emaciated, marantic infants who have been suffering for a long time from inanition. They also

occur in the course of, or immediately after acute gastro-intestinal disturbances. The reason for this is not at all clear. Fairbanks suggests that they are "mostly of nervous origin brought about by a disturbance of the sympathetic nervous system induced by irritants, chiefly toxins, originating in the gastro-intestinal tract." It is assumed that under such conditions there is a diminished resistance of the capillary blood vessels, thereby allowing the extravasation of fluid from the blood stream into the tissues. Courteney and Fales have shown that the blood of such babies is very much diluted. The water content of the blood of normal infants studied by them was 82 per cent. by weight, while in infants suffering from nutritional œdema the water was as high as 87 per cent. Because of this dilution the proportion of salts is lowered, but since no accurate methods exist for estimating the blood volume in young infants, it is not possible to determine whether or not the total quantity of salts in the blood was actually increased during the period of œdema, despite the relative decrease.

Excessive water intake, as for instance by too often repeated hypodermoclysis may give rise to generalized œdema. This is purely mechanical and is due to the inability of the kidneys to excrete the fluids with sufficient rapidity. This is frequently seen in infants with severe intestinal intoxication complicated by acidosis. In such cases it is imperative to establish a free flow of urine as rapidly as possible in order to eliminate the toxic substances causing the acidosis, accordingly fluids are often given in excess. Such an œdema will disappear in a few hours whenever the administration of water is withheld.

Prolonged feeding of a carbohydrate rich diet is commonly followed by the development of œdema. This is not uncommon after a gastro-intestinal upset, when the baby is kept on barley water or sugar solution for too long a period without the addition of sufficient fats and proteins to balance the diet properly. In the more severe forms of this condition there may be associated with the œdema dryness of the skin, rigidity of the muscles, and later, keratomalacia. This condition has been called "starch atrophy".

We have seen œdemas in older children whose diet consisted almost exclusively of cereals. Many theories have been advanced in attempts to explain the relationship between high carbohydrate feeding and dropsy, but so far none of the theories are entirely acceptable.

The occurence of nutritional œdema in an undernourished infant must always be regarded with grave concern. It is always the indication of a very serious metabolic disturbance and generally

indicates a bad prognosis. It is extremely dangerous for a baby with nutritional œdema to lose his œdema too rapidly; for when this occurs, death almost invariably follows. It is not sufficient then merely to change the diet of the baby. It is important that the weight be carefully watched and in the event of a large daily loss, as much as six or seven ounces, to check the rapid water loss by hypodermoclyses of normal saline, despite the presence of the œdema. We have frequently found this a very valuable therapeutic measure and have felt that the hypodermoclyses saved the lives of several of these infants. Transfusions of small quantities of blood when the œdema was at its height has been tried with success in some cases.

Immediate attention must be given to the diet whenever an infant develops nutritional œdema. If the baby has been fed an unbalanced diet, unduly high in carbohydrates, the logical food is protein milk. This food has a high fat and protein content with a low sugar. Although the salts of protein milk are only slightly lower than those of cow's milk, the soluble salts of sodium and potassium are greatly reduced in the process of manufacture. On such a diet the baby promptly begins to lose the œdema. Despite the high sugar content of breast milk, it is the only food that such a baby should have once the œdema has gone. The reasons are, first, that it is the physiological infant food; and, secondly, that the salt content is only 2 gm. per litre. The baby with nutritional œdema is a marantic infant of the worst type and wet nursing offers him his best chance of recovery. The wet nursing should be continued until the nutrition has improved considerably. Not until then may we begin to substitute one feeding at a time, a carefully chosen milk formula. Many other forms of œdema are seen in children, but few of them are of such serious import as the so-called nutritional type. Congenital œdema is sometimes seen, and this condition has been known to be a cause of dystocia. These infants are either stillborn or die shortly after birth. Œdema of the newborn, occurring a few days after birth, is another uncommon condition. This is more frequently seen in premature infants. Localized œdema of an arm or leg, of the eyelids or face has been described coming on irregularly, sometimes after exposure to cold. These are of the angioneurotic type and are frequently hereditary.

Although not very common, there is a form of generalized œdema, not associated with any demonstrable nephritis, coming on after acute infections especially scarlet fever. Albuminuria is not associated with this condition. Children with generalized

eczema are prone to the development of dropsy. Menschikoff has pointed the relation of chloride retention to exudative processes of the skin. The tendency to œdema in these children might be explained on this basis. We have seen a case of generalized eczema with œdema of the feet in a fairly well nourished child. The urine was scanty and concentrated; it contained albumen and casts. With the forcing of water, the œdema promptly disappeared and the urine became normal.

Œdema of the feet often occurs associated with tetany. This is not of very great importance. It usually disappears when the primary condition is treated. Similarly, slight œdema of the feet may sometimes be seen in scurvy, and here, too, requires no special attention other than the treatment of the scurvy itself.

The possibility of the presence of general peritonitis should not be overlooked in a young infant with a distended abdomen, vomiting and œdema of the feet. We have several times seen this condition and have been misled by the absence of fever and abdominal tenderness. The signs of peritonitis in young infants are rarely very definite, and the presence of œdema makes the diagnosis more difficult.

It is important to emphasize the fact that the presence of œdema in infants and young children does not necessarily mean acute nephritis even if albumen and casts are present in the urine. Each case must be gone into very carefully, particularly as regards the previous feeding, and all the causes mentioned above should be taken into consideration. The so-called nutritional œdemas are the most serious types with which we have to deal. Occurring, as they do, in young ill-nourished infants, they must always be regarded very seriously and treated with the greatest care.

RECENT ADDITIONS TO OUR KNOWLEDGE OF INFANTILE SCURVY

By Lionel M. Lindsay, M.D.

Montreal

So much interest has been shown in the subject of scurvy during the past few years and so many new discoveries have been made both in England and America, that a consideration of the results, especially in their relation to pediatrics, may be of profit.

Funk and Hopkins were the first to revive our interest in scurvy by demonstrating the necessity of certain accessory food elements in order to maintain health and promote growth. To these hypothetical elements Funk gave the rather inappropriate name of vitamines.

Since then the influence of certain foods on nutrition has received a great deal of attention, not only with the result that growth and nutrition were found to be dependent on the presence of specific vitamines in the diet, but also that deficiency or absence of them in food may lead to the development of such diseases as scurvy, beri-beri and pellagra, now grouped together under the caption of "deficiency diseases".

Scurvy has been shown to be due to the absence in the diet of vitamines, most abundant in some fresh fruits, green vegetables, and some tubers. It is also present in small amounts in fresh meat and milk, but apparently does not exist in yeast, vegetable fats, and cereals.

This anti-scorbutic vitamine appears to a great extent to be destroyed by prolonged high temperatures, and may be injured by drying and other methods of preservation. Boiled milk, and to a less extent, pasteurized milk, owing to this injurious influence of heat may be deficient in this important element; even the raw milk may contain comparatively little.

The following table compiled from the extensive investigations of the American Pediatric Society, shows at a glance the various forms of milk and milk foods, during the administration of which scurvy developed.

Breast-milk	3 per cent.
Raw cow's milk	1·5 "
Pasteurized milk	5 "
Condensed milk	15 "
Sterilized milk	25 "
Proprietary infant foods	50 "

Recent investigations by McCollum and Simmonds[1] have shown that the vitamine content of milk largely depends on the kind of food consumed by the cow; hence it is easy to understand how raw milk may be more lacking in vitamines in winter when cows are fed on dry fodder than during the summer season when they obtain fresh grass. Scurvy may occasionally occur in babies nursed entirely at the breast, owing possible to the fact that the mother's diet contained no fresh fruit or fresh vegetables. It thus

appears that the mammary gland can only pick up from the blood the essential vitamines and pass them into the milk, but cannot produce them *per se*.

Professor Comby, of Paris, lays it down as an axiom that every infant brought up on sterilized food for several months, to the exclusion of any living fresh articles, is liable to develop scurvy. This has been questioned, and many authorities hold that there must be a predisposition to the disease in an exudative or hæmorrhagic diathesis. We know, however, that scurvy can always be produced in guinea-pigs in from ten to twenty days by feeding them on rolled oats and hay, with the addition of some stimulant to peristalsis, such as ager-ager, mineral oil, or phenolphthalein. It has been also suggested that the young of healthy mothers are born with a reserve supply of vitamines sufficient to protect them until they begin to take other food than milk, and that only those with insufficient reserve will develop scurvy.

Hess states that scurvy requires about six months to develop sufficiently to be recognized clinically, and regards it as probable that in a number of cases the beginning of scurvy may be latent and pass unrecognized. One should always suspect scurvy in an infant, who after thriving for some time, begins to fail, stops gaining, or actually loses, is fretful and refuses food. When, however, a case reaches the florid stage, there can be little difficulty in the diagnosis, for the tenderness and pseudo-paralysis of the limbs, with hæmorrhages about the teeth and blood in the urine are such striking symptoms that few could overlook them. But it must be remembered that hæmorrhages do not occur in the gums unless teeth are present and as scurvy is often associated with rickets and delayed dentition, this symptom is often absent. Frequently the pain and pseudo-paralysis have been attributed to "rheumatism" and real paralysis, and the child is allowed to suffer for weeks and is even tortured with massage, because the true nature of the condition is not recognized. Other symptoms may be misleading. Professor Morse recently gave a clinic at Harvard, on an infant whose chief symptom was marked protrusion of both eyeballs (proptosis) due to hæmorrhage into the sockets. Physicians who had seen the infant previously had failed to recognize the underlying scurvy.

There is no disease so satisfactory to treat as infantile scurvy. Improvement begins immediately anti-scorbutic measures are employed, and acute symptoms may disappear within a week. Treatment consists in simply supplying the necessary vitamines to the

diet. Orange-juice is pre-eminently the ideal anti-scorbutic, but if oranges are too expensive or difficult to obtain, the juice of the swede or the tomato may be employed, which are almost equally efficacious and are well borne by small infants. These juices are usually given in doses of one half to one ounce daily. Less than this amount may possibly be ineffective, in some cases this amount may be doubled. They are best given in one or two doses during the day one hour before feeding, when the stomach contains but little milk. It is also advisable to correct any error in feeding, and change from any patent or dessicated food to one containing some fresh milk, but this is often not essential to the cure.

The prevention of infantile scurvy is an equally simple matter. Provided the mother's diet contains a certain amount of fruit and vegetables, feeding at the breast practically insures the infant against the disease.

Assuming that scurvy takes some months to develop and that every child is endowed with a reserve story of vitamines, we need not have anxiety about its development in an artificially-fed infant until it is three or four months old. After that age it is advisable to add small quantities of an appropriate anti-scorbutic juice every day or two to the infant's dietary. In this way it is possible to correct a slight negative balance of the vitamine principle before the onset of even latent scurvy.

Hess[2] has recently demonstrated the anti-scorbutic property of strained canned tomatoes, both as a preventive and as a remedy, and has adopted its employment in one of the infants' asylums of New York as a routine, in place of orange-juice, with the best results, thus saving hundreds of dollars to the institution. To the same observer we also owe another observation of economic importance, viz.: that a watery extract of grated orange peel may be used to eke out the quantity of orange juice. He also found that orange juice could be boiled without apparently losing much of its value if used immediately afterwards. Still more recently Givens and McClugage[3] claim that dessicated orange juice will retain most, if not all, of its anti-scorbutic vitamines. Similarly, young fresh vegetables may be dehydrated and still retain their anti-scorbutic potency, but old or stale vegetables will lose this principle to a large extent.

An interesting experiment carried out by Hess was the intravenous administration of boiled orange-juice rendered slightly alkaline by sodium hydroxide. This was followed by the almost immediate disappearance of all acute symptoms of scurvy in an

infant. Harden and Zilva[4], however, have shown that orange juice will lose its anti-scorbutic power if made alkaline with NaOH and allowed to stand over-night before being given by mouth. Hence the length of time the food is exposed to the deleterious influence of either alkali or heat is of great importance in the effect produced upon the anti-scorbutic principle.

In order to ascertain what foods contain valuable amounts of anti-scorbutic vitamines, Chick, Hume, Rhodes[5] and others working at the Lister Institute, have carried out a great number of experiments ou guinea pigs and monkeys, the only known animals apart from man that are susceptible to scurvy. Although the guinea pig is more susceptible to scurvy than either man or monkey, there is every reason to believe the disease is identical in all, and that the relative value of different anti-scorbutics is the same for all.

Various foods were tested by them both qualitatively and quantitatively on these animals, and many important observations made. It was found that fresh orange juice is approximately four times as potent as fresh lime juice; the value of preserved lime juice (as rationed to the navy) was practically nil. It was also found that fresh grapes were of little anti-scorbutic value. Of the raw vegetable juices examined that of the swede proved to be by far the most potent; carrots were much inferior and beets practically useless. The potato, even when cooked, has definite anti-scorbutic properties, and may be given to infants as "potato water", made by adding one tablespoonful of baked mashed potato to a pint of water. This may be used as a diluent for the milk as it possesses the same colloidal action as barley water.

Swede juice was a great boon to the English Infant Welfare Centres during the late war and was used with great success.

As regards animal foods, it is interesting to note that raw meat and meat juice are not rich in water-soluble and anti-scorbutic vitamines, but glandular tissues, like pancreas, liver and kidney, have considerable value. Heart muscle stands between these two groups in this respect.

The anti-scorbutic value of cow's milk, fresh, heated and dried, has been investigated by Barnes and Hume[6]. Cow's milk, even when fresh, was found to be comparatively poor in anti-scorbutic properties, while dried milk was found to be of still less value. Scalded milk stood in an intermediate position. Furthermore, winter milk seems to be inferior to summer milk corresponding to the differences in the cow's diet during these seasons.

In opposition to these conclusions, Dr. Coutts states in the report of the British Local Government Board that in the process of drying, milk loses none of its anti-scorbutic property. Hess, moreover, states that he was able to cure scurvy both in infants and in guinea pigs by feeding a certain brand of dried milk.

It would therefore appear that all dried milks cannot be regarded as absolutely lacking in this principle. The amount of destruction of the vitamine that takes place depends on the process of drying employed. The shortness of the time the milk is subjected to heat, and the rapidity of the dessication seems to occasion a minimum degree of destruction.

A knowledge of these facts is of great importance these days, when condensed milk and patent foods are so largely used in infant feeding, and when some pediatrists advocate boiled milk mixtures. The physician must not only see to it that no symptoms of scurvy develop, but also that the health and nutrition of the infant are not impaired by the lack of so simple a precaution as the administration of orange juice, potato-water or canned tomato.

References:

1. McCollum and Simmonds—"The Nursing Mother as a Factor of Safety in the Nutrition of the Young." *Amer. Jour. Physiol.*, 46/3/1918.
2. Hess and Unger—"Canned Tomatoes as an Anti-scorbutic." *Proc. Soc. Exp. Biol.*, 16/1/1918.
3. Givens and McClugage—"The Anti-scorbutic Property of Fruits." "An experimental Study of Dried Orange Juice." *Amer. Jour. Dis. Child.*, July, 1919.
4. Harden and Zilva.—"The Susceptibility of the Anti-scorbutic Principle to Alkalinity." *Lancet*, September 7th, 1918. "The Anti-scorbutic Factor of Lemon Juice." *Lancet*, January 4th, 1919.
5. Chick *et al.*—"The Anti-scorbutic Principle in Limes and Lemons." *Lancet*, 2/735/1918. "The Anti-scorbutic Value of the Raw Juice of Root Vegetables." *Lancet*, 2/774/1918.
6. Barnes and Hume.—"The Anti-scorbutic Properties of Fresh, Heated and Dried Cow's Milk." *Lancet*, August 23rd, 1919.

Obituary

ROBERT A STEVENSON, M.D.

There died in November last, in Toronto, a notable graduate of McGill University of the time of Osler, and one of his oldest friends. A graduate of 1871, he was of the class a year ahead of Osler. After graduation, Dr. Stevenson practised for some years at Strathroy, Ontario, and in 1874 went abroad for a year. The writer, who knew him at college, renewed his acquaintance in London, and spent some months with him in Edinburgh following

Lister, who at that time was operating under the spray. He was a most charming companion, well read, intelligent and with great facility of expression. He had a wide knowledge of many subjects outside of medicine and would have made a great success as a teacher. He had a very logical mind, acute powers of observation, a winning manner and handsome presence with a good vocabulary and fluency of speech—all the qualities for a good teacher of clinical medicine. But although at one time he thought of this career, circumstances prevented him taking it up. A good many years ago he settled in Toronto as a general practitioner and was most successful, having soon secured a good practice amonst the best people. He was an expert anæsthetist and was much sought after for his skill in this branch of the profession.

He was a son of Judge Stevenson of Cayuga, Ont., his mother was a direct descendant of the famous Colonel Butler of Butler's Rangers, so famous during the war of the Revolution. He is survived by his daughter who is married to General Cartwright, C.B., R.E. A long and painful illness preceded his death, but during the whole of this period he was always intensely interested in everything new in medicine. The war and all outside affairs also interested him greatly and he was always ready to discuss affairs in general with his many friends. He contributed little to medical literature and was not prominent in medical societies, but, notwithstanding, his opinion in difficult cases was much sought after. He was well read, cultured and courteous and belonged to a type of medical men which is rapidly passing away. F. J. S.

Dr. William A. Molson, one of the oldest of the present-day practitioners of Montreal, died of cardio-renal disease on January 4th, after a prolonged illness. Dr. Molson was co-editor with the late Dr. George Ross of the old *Canada Medical and Surgical Journal* during the years 1879 to 1882, at the period when Sir William Osler was one of its most prolific contributors. He was one of the attending physicians to the Montreal General Hospital for over a quarter of a century. The early years of his service coincided with the appointment of Sir William Osler as pathologist to that institution, and Dr. Molson was both active and successful in obtaining autopsies upon the patients who died under his care for the benefit of his great confrère, and his name is frequently mentioned in Osler's "Practice of Medicine" in connection with original cases quoted by its author. Dr. Molson, in his later years, was best known for his charitable work among the poor, for whom, in his large general practice, he did an extensive and valuable work.